ACUTE KIDNEY INJURY IN ALLOGENIC HAEMATOPOETIC STEM CELL TRANSPLANTATION

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BONAFIDE CERTIFICATE

This is to certify that the work presented in this dissertation titled "**Acute Kidney Injury in Haematopoetic Stem Cell Transplantation**" done towards fulfillment of the requirements of the **Tamil Nadu Dr. M.G.R. Medical University, Chennai for the D.M. (Branch–III) (Nephrology)** exams to be conducted in August 2013, is a bonafide work of Dr Venu Madhav Gowrugari ,done under my guidance and supervision. This dissertation has not been submitted, fully or in part to any other board or University.

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CONTENTS

ABBREVIATIONS

AKI- Acute Kidney Injury

AKIN- Acute Kidney Injury Network,

RIFLE- Risk injury Failure Loss End stage renal disease

AML- acute myeloid leukemia,

ALL- acute lymphoid leukemia,

CML-Chronic myeloid leukemia

CNI- Calcineurin inhibitor,

GVHD-Graft versus host disease,

AGE-Acute gastroenteritis

TBI-Total Body Irradiation,

GVHD-Graft versus host disease,

SOS-Sinusoidal occlusive disease,

CAD-coronary artery disease,

MUD-Matched unrelated donor,

TBI-Total Body Irradiation

NMDP (U.S)-National Marrow Donor Program me

DKMS (Germany)-Deutsche Knochenmarkspenderdatei

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Clinical profile of acute kidney injury (AKI) in allogenic hematopoietic stem cell transplant (HSCT) recipients

Introduction and Aim: Acute kidney injury is a common complication after allogenic hematopoietic stem cell transplantation (HSCT). Different studies have shown varied incidences. Aim of study was to identify the incidence and outcomes of acute kidney injury associated with allogenic hematopoietic stem cell transplant (HSCT).

Methods: 203 HSCT recipients at Christian Medical College, Vellore from Jan 2010 to Dec 2011 were studied and followed up to 12 months from the time of allogenic HSCT. Data was collected from HSCT database and Clinical workstation network. AKIN and RIFLE criteria of AKI were used to define acute kidney injury. Mortality and chronic kidney disease were primary outcome variables studied.

Results: The 203 allogenic HSCT recipients (172 matched related and 31 matched unrelated donors, M: F = 1.57:1; mean age 22 ± 14.5 years), had a total of 170 episodes of AKI by AKIN criteria. A minimum of one episode of AKI was observed by RIFLE criteria in 140 (69%) and AKIN criteria in 149(72.9%) patients. There was good agreement between the RIFLE (R-34.5%, I-24.1%, F-10.3%) and AKIN classification (Stage 1- 38.4%, 2- 24.6%, 3- 10.3%) (K=0.75). AKI occurred at a median of 17 (2-290) days after the HSCT. Common causes of AKI were sepsis/SIRS (34.5%), nephrotoxic antibiotic use (6.4%), Cyclosporine toxicity (18.2%) and dehydration (eg. - gastroenteritis, GVHD in GI tract, 3.0%). Mortality was observed in 29.5% at the end of 1 year. SOS, Amphotericin, Sepsis, Systemic mycosis are independent risk factor for mortality. AKI is a significant risk factor for mortality (84.2% vs. 67.6%. $p<0.011$). Among survivors, chronic kidney disease was noted in 41.0% of patients at the end of follow up. Kaplan Meier survival analysis of mortality with severity of renal dysfunction demonstrates a stepwise incremental risk according to the AKIN classification ($p<0.001$).

Conclusions: AKI is very common after allogenic HSCT and accounts for significant mortality and morbidity among these patients. AKIN classification of AKI has prognostic significance among allogenic HSCT patients in a follow up of 1 year.

ACUTE KIDNEY INJURY (AKI)

INTRODUCTION:

Acute Kidney Injury (AKI) is defined as an abrupt loss of renal function that causes retention of urea and nitrogenous waste products and in deregulation of extracellular volume and electrolytes.

ADQI group proposed a consensus definition, called RIFLE criteria [1]. A modification of the RIFLE criteria was subsequently proposed by Acute Kidney Injury Network (AKIN)

[2-5].

RIFLE CRITERIA —

The RIFLE criteria consists of three levels of injury (Risk, Injury, and Failure) based on either degree of rise in serum creatinine or urine output, and two outcome measures (Loss and End-stage renal disease).

***Table 1: The RIFLE classification** [1]:*

Risk	SCr increased × 1.5	UO < 0.5 mL/kg/h × 6 h	GFR decreased >25%
Injury	SCr increased × 2.0	UO < 0.5 mL/kg/h × 12 h	GFR decreased >50%
Failure	SCr increased × 3.0 Or SCr ≥4 mg/dL; acute rise ≥0.5 mg/dL	UO < 0.3 mL/kg/h × 24 h or anuria for 12 hours	GFR decreased >75%
Loss	Persistent acute renal failure: complete loss of kidney function >4 wk		
ESRD	Complete loss of kidney function >3 months		

The RIFLE criteria correlated with prognosis in a number of studies [5-11]. A systematic review of several studies demonstrated a stepwise increase in relative risk of death in patients who met RIFLE criteria for various stages of AKI [11)

The reference to change in GFR is not included in more recent AKIN classification system.

AKIN CRITERIA —

The modification of RIFLE criteria was proposed by Acute Kidney Injury Network (AKIN). The AKIN proposed both diagnostic criteria for AKI and a staging system that was based on the RIFLE criteria [2-4].

Diagnostic criteria — Diagnostic criteria for ARF are an acute (within 48 hours) rise serum creatinine concentration of ≥0.3 mg/dL (26.4 micro mol/L) from the baseline, an increase in serum creatinine of ≥50 %, or oliguria <0.5 mL/kg/hr for >6 hours.

The last two of these criteria are similar to RIFLE "risk" criteria. The addition of an absolute rise in serum creatinine of ≥0.3 mg/dL is based on epidemiologic studies that have demonstrated an 80 percent increase in mortality risk associated with changes in serum creatinine concentration of as small as 0.3 to 0.5 mg/dL [12]. Including a time limit of 48 hours is based upon data that showed that poorer outcomes with small changes in the creatinine when the rise in creatinine was observed between 24 to 48 hours [13,14].

Two additional caveats were proposed by the AKIN group:

- The diagnostic criteria should be applied only after volume status is corrected.
- Urinary tract obstruction should to be excluded, if urine output was used as only diagnostic criteria.

Staging system — the classification or staging system for AKI is consists of three stages of AKI with increasing severity, which corresponds to RIFLE criteria.

Risk =stage 1

Injury = stage 2

Failure = stage 3

Loss and ESRD are removed from the staging system and defined as outcomes.

The clinical applicability of the above staging systems is not certain. However, they have some utility in standardizing the definitions for epidemiologic studies and for establishing inclusion criteria and endpoints for various clinical trials.

Ultimately these definitions are more likely to be replaced by more sensitive and specific biomarkers of renal injury in future.

KDIGO MODIFICATIONS TO RIFLE AND AKIN —

The KDIGO clinical practice guidelines revised definition of AKI while retaining the AKIN staging criteria [15]. In the KDIGO definition, the time frame for a absolute increase in serum creatinine of 0.3 mg/dL is retained from AKIN definition (48 hours) while the time frame for a >50 % rise in serum creatinine is the 7 days, as was originally suggested by the RIFLE criteria. The KDIGO criteria only utilizes changes in serum creatinine and urine output ,but not changes in GFR for staging, with the exception of children <18 years, for whom an acute decrease in estimated GFR to <35 ml/min per 1.73m2 is included in the criteria for stage 3 AKI. As with the RIFLE and AKIN staging systems, patients should be classified according to criteria that result in the highest (ie, most severe) stage of injury.

Table 2: Using the KDIGO criteria, AKI staging:

Stage 1	1.5-1.9 times baseline OR ≥0.3 mg/dl (≥26.5 mmol/l) increase in the serum creatinine	Urine output <0.5 ml/kg per hour for 6 to 12 hours.
Stage 2	2.0-2.9 times baseline increase in serum creatinine	Urine output <0.5 ml/kg per hour for ≥12 hours.
Stage 3	3.0 times baseline increase in serum creatinine OR increase in serum creatinine to ≥4.0 mg/dl (≥353.6 micromol/l)	Urine output of <0.3 ml/kg per hour for ≥24 hours, OR anuria for ≥12 hours OR the initiation of renal replacement therapy

PATHOPHYSIOLOGY —

AKI is characterized by an abrupt decline in renal function. The aetiology is classified into pre renal, intrinsic renal and post renal causes.

Prerenal AKI —

Decreased kidney function due to prerenal disease occurs in two settings (16)

- When renal ischemia is part of a generalized decrease in tissue perfusion.
- When there is selective renal ischemia.

Systemic hypoperfusion is initially sensed by cardiac and arterial receptors that respond to changes in pressure (or stretch). When the mean arterial pressure is reduced, due to a reduction in either in cardiac output or systemic vascular resistance, activation of these receptors increases sympathetic neural tone and the release of both renin (leading to the generation of angiotensin II) and antidiuretic hormone. The ensuing arteriolar and venular constriction and stimulation of cardiac function return the systemic blood pressure and cardiac output toward normal. The arteriolar vasoconstriction occurs primarily in the renal, splanchnic, and musculocutaneous circulations, resulting in the relative preservation of blood flow to the heart and brain.

Although these are appropriate systemic responses, renal vasoconstriction can diminish renal blood flow and the glomerular filtration rate (GFR), which is flow-dependent. In addition, if the compensatory systemic responses are incomplete, persistent decrease in cardiac output and/or arterial pressure can contribute to the decline in GFR.

A common cause of prerenal disease is true volume depletion, which includes hypovolemia caused by dehydration, hemorrhage, or renal (diuretics) or gastrointestinal (vomiting, diarrhoea) fluid loss. Renal perfusion can also be reduced in edematous states such as heart failure and cirrhosis due to myocardial dysfunction and splanchnic venous pooling and systemic vasodilation, respectively.

With all of these processes that cause prerenal disease, the GFR is diminished because of decreased renal blood flow. The glomeruli, kidney tubules, and interstitium are intact. The

appropriate treatment is to increase renal perfusion, as with volume repletion in patients with true volume depletion.

Acute tubular necrosis (ATN) —

With prolonged and/or severe ischemia, ATN can occur. This can result in histologic changes, including necrosis and denudation of the tubular epithelium with occlusion of the tubular lumen by casts and cell debris.

However, this injury pattern is not universal and the pathophysiologic mechanisms may be different in certain disease conditions. As an example, sepsis-associated high cardiac output failure is associated with dilatation of both afferent and efferent arterioles, which will reduce renovascular resistance and tend to maintain renal blood flow. However, the efferent arterioles usually dilate to a greater degree, which can lead to a decrease in intraglomerular pressure and therefore in GFR. Most of the evidence is derived from animal data [17,18].

Any of the processes associated with prerenal disease can cause ATN, but kidney damage most commonly occurs in patients with hypotension, particularly in the settings of surgery, sepsis, or obstetrical complications. The other major causes of ATN include a variety of nephrotoxins that directly damage renal tubules via a number of different mechanisms [19].

Although renal ischemia is the most common cause of ATN, the sensitivity of individual patients to a decrease in renal perfusion is variable. In some patients, a few minutes of hypotension is sufficient to induce ATN, whereas others are able to tolerate hours of renal ischemia without structural damage to the kidney, displaying the findings of prerenal disease such as a normal urinalysis and a low fractional excretion of sodium. Such patients can eventually develop ATN if renal perfusion is not improved.

ETIOLOGY — Both prerenal disease and ATN can occur in a variety of settings. In addition, prerenal disease, if severe, is a common cause of ATN.

Causes of prerenal disease — prerenal disease may result from the following:

- **True volume depletion** — Volume depletion may be caused by, renal losses (diuretics, glucose induced osmotic diuresis), gastrointestinal disease (diarrhoea, vomiting, bleeding), skin or respiratory losses (insensible fluid losses, sweat, burns), and third space sequestration (skeletal fracture or crush injury).
- **Hypotension** — severely decreased blood pressure can result from shock (hypovolemic, myocardial, or septic) and posttreatment of severe hypertension.
- **Edematous states** — Heart failure and cirrhosis can result in marked reductions in kidney perfusion that parallel the severity of the underlying disease. The respective mechanisms are decreased cardiac output in heart failure and splanchnic venous pooling and systemic vasodilation in cirrhosis Nephrotic syndrome, mostly in adults with minimal change disease, can also lead to AKI. Decreased renal perfusion, reduced glomerular permeability, and excessive diuresis are among the mechanisms that may contribute to AKI.
- **Selective renal ischemia** — Bilateral renal artery stenosis or unilateral renal artery stenosis in a solitary functioning kidney which is worsened by treatment with angiotensin converting enzyme inhibitors (ACEI), angiotensin II receptor blockers(ARB), or direct renal inhibitors.
- **Drugs affecting glomerular hemodynamics** — Drugs that affect glomerular hemodynamics can reduce the glomerular filtration rate by lowering the intraglomerular pressure that drives this process. This can occur by decreasing either afferent (preglomerular) arteriolar dilatation (eg, with nonsteroidal antiinflammatory drugs or calcineurin inhibitors) or efferent (postglomerular) arteriolar constriction (eg, with ACEI OR ARB).

The effect of nonsteroidal anti inflammatory drugs (NSAIDS) is primarily observed in patients with underlying renal hypoperfusion due to true volume depletion, heart failure, or cirrhosis in which prostaglandin synthesis within or near the glomerulus is increased by vasoconstrictors such as angiotensin II and norepinephrine and vasodilator prostaglandins help preserve renal reperfusion and glomerular filtration. The effect of angiotensin inhibitors is also seen in these settings since angiotensin II helps maintain the intraglomerular pressure and glomerular filtration rate by increasing efferent arteriolar

resistance. The mechanism with calcineurin inhibitors is less clear. The supporting data are discussed in detail elsewhere.

Causes of ATN — there are three major causes of ATN: renal ischemia; sepsis; and nephrotoxins.

Renal ischemia — All causes of severe prerenal disease, particularly if accompanied by hypotension, surgery, and/or sepsis, can cause postischemic (also called ischemic) ATN. For reasons that are not well understood, ATN is an **unusual** complication of heart failure and it is not clear if prolonged renal ischemia alone can lead to ATN in patients with cirrhosis in the absence of some other risk factor (eg, hypotension due to bleeding, or aminoglycoside therapy).

Sepsis — Sepsis-induced ATN is often associated with prerenal factors such as decreased renal perfusion and systemic hypotension. Other factors can also contribute, including release of cytokines and activation of neutrophils by cytokines.

Nephrotoxins — A number of drugs and exogenous and endogenous toxins can cause ATN [19]. These include:

- Aminoglycosides
- Heme pigments
- Cisplatin
- Radiocontrast media
- Pentamidine
- Foscarnet
- Cidofovir
- Tenofovir [20-22]
- Intravenous immunoglobulin, mostly in products containing sucrose which is thought to be taken up by tubular cells, leading to cell swelling and tubular obstruction
- Mannitol, primarily in patients treated with more than 200 to 300 g/day
- Hetastarch (also called hydroxyethyl starch), a hyperoncotic colloid used for fluid resuscitation in intensive care units [23-25]. The risk of AKI appears to be less with low volume therapy [26].

FREQUENCY OF PRERENAL DISEASE AND ATN AS A CAUSE OF AKI — AKI, as defined by RIFLE criteria, has been observed in 9 percent of hospitalized patients [27], and in more than 50 percent of patients in the intensive care units [28]. Approximately 65 to 75 percent of cases of AKI in a hospital are because of either prerenal AKI or ATN [29-32]. A study from Spain, for example, evaluated causes of AKI [30].

Table 3: The most frequent causes of AKI [30]

ATN	45 %
Prerenal disease	21 %
Acute on chronic renal failure	13 %
Urinary tract obstruction (UTO)	10 %
Glomerulonephritis or vasculitis	4 %
Acute interstitial nephritis (AIN)	2 %
Atheroembolic disease	1 %

Another study based upon data from the Program to Improve Care in Acute Renal Disease (PICARD) examined the aetiology of AKI in 618 critically ill patients in ICU in five medical centres in the United States [32]. The most common causes were postischemic ATN (primarily due to sepsis or hypotension and accounting for 50 percent of cases), prerenal disease (hypovolemia, hemorrhage, heart failure, shock, hepatorenal syndrome), nephrotoxicity (radiocontrast media and rhabdomyolysis were the most common), cardiovascular disease (heart failure, shock), liver disease (hepatorenal syndrome), and multifactorial etiologies.

EVALUATION AND DIAGNOSIS —

The initial step in the evaluation of patients with AKI is a meticulous history and physical examination. When appropriate, prerenal disease must be distinguished from post ischemic ATN.

History and physical examination — this can frequently identify preceding insults or disease processes those results in decreased tissue perfusion that can lead to prerenal disease or post ischemic ATN:

- The history may reveal a cause of decreased tissue perfusion (eg, vomiting, diarrhea, bleeding, or sepsis). In addition, in hospitalized patients, a close examination of the clinical setting may help identify the underlying cause of AKI (eg, hypotension, sepsis, intraoperative events, aminoglycoside therapy, or the administration of radiocontrast media, particularly in patients at increased risk).
- Among patients who develop AKI in the hospital, the day of onset can be determined if the serum creatinine concentration has been measured daily. Suppose, for example, that a patient has had a stable serum creatinine concentration, which began to rise progressively on day five. In such a patient, there must have been some insult on day four (eg, hypotension, radiocontrast media) or a cumulative insult that has become clinically apparent (eg, aminoglycoside therapy). Careful perusal of the patient's chart may identify the probable cause.
- Findings on physical examination may suggest hypovolemia such as otherwise unexplained tachycardia, dry mucous membranes, decreased skin turgor, cool extremities, supine and/or orthostatic hypotension, and, particularly in the elderly, sunken eyes. Heart failure and cirrhosis can result in edema, ascites, and other signs of specific organ dysfunction. Abdominal distension leading to intraabdominal hypertension and abdominal compartment syndrome may be a complication of abdominal surgery. The mechanisms responsible for decreased renal perfusion are discussed elsewhere.

Distinction of prerenal disease from ATN — Distinguishing ATN from prerenal disease should be considered in patients who have a suggestive history and physical examination (as described in the preceding section) and no evidence for another cause of AKI such as

aminoglycoside therapy, glomerulonephritis (which is typically associated with hematuria and dysmorphic red cells with or without red cell casts, and variable degrees of proteinuria), acute interstitial nephritis (which is often drug-induced and typically associated with pyuria with or without white cell casts or hematuria but **not** red cell casts), and urinary tract obstruction (which is diagnosed by imaging studies).

There are three major diagnostic approaches that, in the appropriate clinical setting, are used to distinguish prerenal disease from ATN and from other causes of AKI [33-37]:

- Urinalysis.
- Fractional excretion of sodium and, to a lesser degree, the urine sodium concentration. The fractional excretion of urea may be helpful in patients being treated with diuretics.
- Response to fluid repletion in patients who have evidence of volume depletion, which is the gold standard for the diagnosis prerenal disease. This does not apply to prerenal disease due to heart failure (cardiorenal syndrome) or cirrhosis (hepatorenal syndrome).

Some patients have an intermediate syndrome with features of both prerenal disease and ATN. The relative contribution of ATN can be assessed by evaluating the response to fluid repletion.

Other parameters that may be helpful in selected patients include:

- Rate of rise of serum creatinine concentration.
- Blood urea nitrogen (BUN)/serum creatinine ratio.
- Urine osmolality.
- Urine volume.

In patients with prerenal acute AKI due to cardiorenal syndrome or abdominal compartment syndrome, definitive diagnosis should be made with cardiac functional evaluation

(eg, cardiac echo, invasive hemodynamic monitoring) or transduced bladder pressure, respectively.

Table 4: Renal indices

Diagnostic Index	Prerenal AKI	ATN
Fractional Na excretion	<1	>2
Urine Na concentration meq/l	<20	>40
Urine creatinine/plasma Cr	>40	<20
Urine specific gravity mOsm/kg H2O	>1.018	1.010
Urine Osmolality	>500	300
Plasma urea nitrogen/Cr	>20	<10-15
Renal failure index	<1	>1
Urine sediment	Hyaline casts	Muddy brown casts

Urinalysis — the urinalysis with sediment examination by urine microscopy is normal in prerenal disease unless it is superimposed on another cause of renal disease. The classic urinalysis in ATN is the presence of muddy brown granular, epithelial cell casts and free renal tubular epithelial cells. In comparison, Ischemic or toxic injury causes tubular cell death or because defective cell-to-cell or cell-to-basement membrane adhesion leads to cell sloughing into the tubular lumen.

The absence of these urinary findings does **not** exclude ATN and their presence does not always establish the diagnosis of ATN as illustrated by the following examples:

- Tubular epithelial cell sloughing and cast formation may not be prominent in patients with less severe disease or nonoliguric ATN, a setting in which the urinalysis may be relatively normal [38].

- Marked Hyperbilirubibemia per se can lead to the formation of granular and epithelial cell casts in the absence of an overt renal tubular injury [39]. In this condition, the urinalysis may **not** differentiate between prerenal disease and ATN. In contrast, marked hyperbilirubinemia does not impair sodium reabsorption; thus, measurement of fractional excretion of sodium (FENa) and the urine sodium concentration remain useful.

Fractional excretion of sodium and urine sodium concentration — the urine sodium concentration is widely used in patients with suspected volume depletion. In the absence of a sodium-wasting state, the urine sodium concentration in hypovolemic states should be less than 20 meq/L and may be as low as 1 meq/L in laboratories able to detect such a low level. However, for the reasons described in the following discussion, measurement of the fractional excretion of sodium is the preferred test for distinguishing prerenal disease from ATN as the cause of AKI.

The urine sodium concentration may be low in prerenal disease (<20 meq/L) in an attempt to conserve sodium, and high in ATN (more than 40 to 50 meq/L) due, in part, to impaired tubular function induced by the tubular injury [33].

The FENa, which includes the urine sodium concentration, is the better test in patients with AKI since it only measures sodium handling (the fraction of the filtered sodium load that is excreted). It is not affected by changes in urine output since the urine volume is **not** included in the formula.

$$\text{FENa, percent} = \frac{\text{UNa} \times \text{SCr}}{\text{SNa} \times \text{UCr}} \times 100$$

Among patients with AKI, the FENa is typically < 1 percent in prerenal disease which is indicative of sodium retention and > 2 percent in ATN [40]. There are conditions in which this distinction may not be accurate, such as ATN superimposed upon a chronic prerenal state such as cirrhosis, a condition in which the FENa may remain below 1 percent.

Limitations of FENa — There are several limitations to the use of FENa in distinguishing prerenal disease from ATN as the cause of AKI. The FENa may remain below 1 percent when

ATN is superimposed upon a chronic prerenal conditions like heart failure or cirrhosis , or in a minority of patients with nonoliguric postischemic (ischemic) ATN who may have persistent renal ischemia and less severe ATN.

- A FENa below 1 percent is not confined to prerenal disease, as it is also associated with other causes of AKI with a low GFR and relatively intact tubular function. These include acute glomerulonephritis, vasculitis, and contrast-induced nephropathy.
- Patients with AKI are often treated with diuretics which, if effective, can have increased FENa, even with prerenal disease. Potential alternative in such patients, which have been less well studied than FENa, include the fractional excretion of molecules such as urea, lithium, and uric acid that are primarily reabsorbed and, with uric acid, secreted in the proximal tubule, which is proximal to the sites of action of loop diuretics (loop of Henle) or thiazide diuretics (distal tubule).
- The FENa defining sodium retention **varies with the filtered sodium load**: below 1 percent when the filtered sodium load is low due to the marked reduction in glomerular filtration rate (GFR) in AKI; and progressively lower as the filtered sodium load increases, reaching a value of **less than 0.1 percent** when the GFR is normal.

Response to fluid repletion — it is a gold standard to distinguish between prerenal AKI secondary to volume depletion and postischemic or nephrotoxic ATN . If sufficient fluid is given to reverse signs of volume depletion (eg, hypotension, cool extremities, low FENa and urine sodium concentration), return of the serum creatinine to the previous baseline within 1 to 3 days is indicates correction of prerenal AKI, whereas persistent AKI is considered to represent ATN.

Although prerenal disease and postischemic ATN are usually discrete entities, some patients have an intermediate presentation with overlap between features of both prerenal disease (eg, low fractional excretion of sodium) and ATN, such as coarse granular casts and/or renal tubular epithelial cells on urine microscopy [36,37,41] and urinary biomarkers of renal tubular injury [42].

In contrast to the prompt recovery to baseline kidney function following volume repletion in pure prerenal disease, patients with prerenal disease who also have renal features of ATN

may have delayed recovery to baseline kidney function after fluid repletion [43]. This may reflect a pattern of patchy tubular injury (accounting for the urine manifestations and delayed recovery) interspersed among normal functioning nephrons [45].

Unless contraindicated, a trial of intravenous fluid replenish is warranted in patients with a clinical history of fluid loss (such as diarrhea, vomiting, or bleeding), and a physical examination consistent with hypovolemia (such as otherwise unexplained tachycardia, decreased skin turgor, cool extremities, and/or supine or orthostatic hypotension). In contrast, fluid administration is **not** warranted and may be harmful (eg, fluid overload and pulmonary congestion) in critically ill patients with AKI who do not have a history or physical or laboratory findings suggestive of hypovolemia [43,44].

Fluid infusion to reverse prerenal disease is generally **not performed** in patients with volume overload state as in cirrhosis or heart failure, particularly in patients with pulmonary vascular congestion. However, there are some settings in which fluid repletion may be considered, particularly in patients with cirrhosis and ascites who do **not** have peripheral edema. In the presence of peripheral edema, the edema fluid can be rapidly mobilized with fluid loss (as with diuretic therapy), which will minimize the degree of intravascular volume depletion [44]. In contrast, in patients with ascites but **no edema**, the ascites fluid can only be slowly mobilized (400 to 500 mL/day). Thus, fluid loss as with excessive diuretic therapy can result in volume depletion and elevations in blood urea nitrogen and serum creatinine. Fluid repletion is typically initiated with intravenous isotonic saline. The rate of fluid repletion varies with the severity of the hypovolemia:

- Patients with severe hypovolemia or hypovolemic shock are typically treated with an initial infusion of **one to two liters of isotonic saline given as rapidly as possible**. The rate of further fluid repletion is governed by the blood pressure response and other clinical signs such as peripheral perfusion, mental status, and urine output. Patients with persistent hypotension are continued at the initial rapid rate as long as there are no signs of volume overload or some other cause of hypotension (eg, sepsis).
- Patients with mild to moderate hypovolemia are treated with isotonic saline at a slower rate. The goal of therapy, volume repletion, requires that the rate of fluid administration be greater than the rate of ongoing fluid losses. One regimen that has

been used is a fluid administration rate that is **50 to 100 mL/h above ongoing fluid losses**. These include insensible losses (usually 30 to 50 mL/h) plus any other fluid losses (eg, gastrointestinal losses).

- The choice of replacement fluid may be influenced by concurrent abnormalities in serum sodium or potassium or the presence of metabolic acidosis. Potassium or bicarbonate may be added in patients who have hypokalemia or metabolic acidosis, respectively, while patients who are hypernatremic may be treated with one-half to one-quarter isotonic saline at two to four times the rate of isotonic saline therapy, respectively, to achieve the same degree of sodium repletion while also treating the hypernatremia. The choice of initial replacement fluid in hypernatremic patients is dependent upon the degree of hypernatremia. Careful monitoring of the rate of correction of the hypernatremia is important with the composition and/or the rate of administration of the replacement fluid being adjusted accordingly.

The following responses to fluid repletion are compatible with prerenal disease: improvement in urine output, restoration of renal function, and a rise in urine sodium concentration which, if it is an isolated finding, can also reflect progression of prerenal disease to ATN. If the urine output and renal function fails to improve with the restoration of intravascular volume and prerenal disease can still be suspected but invasive monitoring is required to correctly assess the patient's volume status and to help guide further therapy.

Other tests that may be helpful

Blood urea nitrogen/serum creatinine ratio — a ratio of 10 to 15:1 observed in ATN , but is commonly greater than 20:1 in prerenal AKI as there an increase in passive reabsorption of urea ,that follows enhanced proximal tubular reabsorption of sodium and water . So, a high ratio is suggestive of prerenal disease with exceptions like, for example:

- BUN will increase out of proportion to serum creatinine: gastrointestinal bleeding [46], tissue breakdown, or glucocorticoid therapy.
- The BUN/serum creatinine ratio often can exceed 20:1 when loss of muscle mass in a elderly or chronically ill patient.

Urine osmolality — Loss of concentrating ability is an early and AN almost universal finding in ATN .The urine osmolality is usually below 350 mosmol/kg . In contrast, a urine osmolality of > 500 mosmol/kg is suggestive of prerenal disease [33]. However, lower values as those in ATN can be seen in prerenal disease.

Urine volume — urine volume is typically, but not always, low (oliguria) in prerenal AKI due to appropriate increases in both sodium and water reabsorption, which limits any further fluid loss. One exception is effective diuretic therapy, which will acutely raise the urine output**.** In comparison; patients with ATN can be either oliguric or nonoliguric.

Investigational biomarkers — although estimation of serum creatinine concentration is widely used for the detection of AKI, it does not permit early diagnosis of ATN since tubular injury can precede a significant rise in serum creatinine. Investigational biomarkers have been evaluated in patients with possible ATN in an attempt to detect tubular injury at an earlier stage but none of them have received widespread approval.

HAEMATOPOIETIC STEM CELL TRANSPLANTATION

Haematopoietic stem cell transplantation was first performed as early as in 1939, when human bone marrow cells were injected intravenously to a patient with Aplastic anemia but the procedure was a failure.[47,48]

Stem cell transplantation was pioneered at Fred Hutchinson Cancer Research Centre from the 1950s to 1970s under E. Donnall Thomas, whose work was awarded Nobel Prize in Medicine in 1990. His pioneering work was also in graft-versus-host disease (GVHD).

Table 5: IMPORTANT LANDMARKS IN HSCT

1939	HSCT first attempted unsuccessfully in Aplastic anemia
1959	Georges Mathé first to perform European HSCT in on five Yugoslavian nuclear workers who suffered nuclear accident.
1968.	Robert A. Good performed First successful HSCT for nonmalignant condition
1990	E. Donnall Thomas, awared Nobel Prize in Physiology or Medicine for his work on HSCT and GVHD

TYPES OF HSCT — there are two main types

Autologous transplant — in autologous transplantation, patients own hematopoietic stem cells are removed before the high dose chemotherapy or radiation is given. After chemotherapy or radiation is complete, the harvested cells are thawed and returned To patient.

Allogeneic transplant — in allogeneic transplantation, the hematopoietic stem cells come from a donor, ideally a sibiling with a similar genetic makeup.

The choice between the two procedures depends on patient age, underlying disease, donor availability.

Allogenic HSCT constitutes 40% of all transplants performed worldwide and it requires donor and recipient HLA matching. As only about 20-25% of patients have a matched sibling donor, an International registry like NMDP, DKMS and Indian registry like DATRI was created with unrelated donor.[49]

PRODEURE OF HSCT:

BY direct aspiration of bone marrow, with the patient under general or spinal anaesthesia or from the peripheral blood stem cells are harvested. HSCT can be done by treating donor with hematopoietic growth factors, which causes stem cells to proliferate and circulate freely in the peripheral blood. The blood is then subjected to leukopheresis to obtain the cells for transplantation.[50,51,52] Umbilical cord blood can also used for the same[53,54]The actual transplantation process involves intravenous infusion of a stem cell product through a large-bore central venous catheter over 60 to 120 min. The stem cells can "home"the bone marrow cavity to re-establish haematopoiesis over the period of next 2 weeks. [55]

Table 6: Indications of HSCT:

Acquired	**Congenital**

Leukemias
• Acute lymphoblastic leukemia (ALL)
• Acute myeloid leukemia (AML)
• Chronic lymphocytic leukemia (CLL)
• Chronic myelogenous leukemia (CML), accelerated phase or blast crisis
 	
Lymphomas
• Hodgkin's disease
• Non-Hodgkin's lymphoma
• Myelomas
	Lysosomal storage disorders
	
	Mucopolysaccharidoses
	
	Immunodeficiencies
	• T-cell deficiencies
	  • Ataxia telangiectasia
	  • DiGeorge syndrome
	• Combined T- and B-cell deficiencies
	  • Severe combined immunodeficiency (SCID), all types
	• Well-defined syndromes
	  • Wiskott-Aldrich syndrome
	• Phagocyte disorders
	  • Kostmann syndrome
	  • Shwachman-Diamond syndrome

• Multiple myeloma Solid tumor cancers • Neuroblastoma • Desmoplastic small round cell tumor • Ewing's sarcoma • Choriocarcinoma Hematologic disease • Myelodysplasia • Phagocyte disorders • Anemias • Paroxysmal nocturnal hemoglobinuria (PNH; severe aplasia) • Aplastic anemia • Acquired pure red cell aplasia • Myeloproliferative disorders • Polycythemia vera • Essential thrombocytosis • Myelofibrosis Amyloidoses	Hematologic diseases • Hemoglobinopathies • Sickle cell disease • β thalassemia major (Cooley's anemia) Anemias • Aplastic anemia • Diamond-Blackfan anemia • Fanconi anemia Cytopenias • Amegakaryocytic thrombocytopenia • Hemophagocytic syndromes • Hemophagocytic lymphohistiocytosis (HLH)

ALLOGENIC TRANSPLANTATION

Four components of allogenic transplantation —

1. Conditioning,

2. Transplantation,

3. Engraftment,

4. Immunoreconstitution.

CONDITIONING:

The preparative or conditioning regimen is a critical element in HSCT and is initiated 1 week prior to HSCT. The purpose of the preparative regimen is to:

1. Adequate immunosuppression to prevent rejection of the transplanted graft
2. Eradication of the native disease.

This involves delivering maximally tolerated doses of multiple chemotherapeutic agents, with or without total body irradiation (TBI). Novel approaches are being practiced to minimize toxicity. (For example, nonmyeloablative preparative regimens for older patients or those with concurrent medical conditions, other alternative are radiolabeled monoclonal antibodies).

PREPARATIVE REGIMEN INTENSITY DEFINED —

Preparative regimens for HCT have been termed myeloablative, reduced intensity, and nonmyeloablative. Generally accepted definitions of these three types of regimens are as follows :

- Myeloablative regimens — A myeloablative (MA) regimen consists of a single agent or combination of agents expected to destroy the hematopoietic cells in the bone marrow and results in profound pancytopenia within one to three weeks from the time of administration. The resulting pancytopenia is long-lasting, usually irreversible, and in most instances fatal, unless hematopoiesis is restored by infusion of hematopoietic stem cells. Examples include total body irradiation ≥5 Gy in a single dose or busulfan >8 mg/kg orally.
- Nonmyeloablative regimens — A nonmyeloablative (NMA) regimen is one that will cause minimal cytopenia (but significant lymphopenia) by itself and does not require stem cell support. Examples include fludarabine plus cyclophosphamide with or without antithymocyte globulin or total body irradiation ≤2 Gy with or without a purine analog. However, the transplant, when given in this setting usually becomes myeloablative, because the engrafting donor T-cells will eventually eliminate host hematopoietic cells, allowing the establishment of donor hematopoiesis.

- Reduced intensity regimens — Reduced intensity regimens (RIC) are an intermediate category of regimens that do not fit the definition of myeloablative or nonmyeloablative. Such regimens cause cytopenias, which may be prolonged and result in significant morbidity and mortality, and require hematopoietic stem cell support. Regimens generally considered as reduced intensity include ≤9 mg/kg of oral busulfan, or ≤140 mg/m2 of melphalan.

TRANSPLANTATION: COUNTDOWN TO DAY 0

- By convention, pretransplant conditioning days are numbered from day –7 to day 0 (the date of transplantation). The days thereafter transplantation are then numbered upward. This universal system is used to describe the time of events. From day 0 to engraftment, the patient's immunity is reduced, and he/she is susceptible to infection. This vulnerability is partly due to breaks in the natural mucosal and skin barriers secondary to mucositis and the indwelling central venous catheter, but neutropenia also contribute to this risk. Febrile neutropenia, an expected complication requires prompt aggressive treatment with broad-spectrum antibiotics. In addition, all patients routinely receive antiviral prophylaxis.16 Throughout this neutropenic period, the patient is confined to a positive-pressure room equipped with high-efficiency particulate air (HEPA) filters which remains a "industry standard.".[56]

ENGRAFTMENT: STEM CELL FUNCTION BEGINS

- Engraftment is a process wherein the donor cells begin to produce new blood components within the recipient's bone marrow cavity. Engraftment is said to have occurred when absolute neutrophil count exceeds 0.5 × 109/L and platelet count of >20,000 cumm. The patient is supported by blood products until engraftment occurs. Engraftment usually occurs between day +10 and day +20 from transplant.8,9 Failure to engraft (primary graft failure) and subsequent irreversible decline of blood counts (secondary graft failure) are very serious complications but hese conditions develop in < 5% of recipients, and they are particularly rare with HLA matched-sibling transplantation.[57]

IMMUNORECONSTITUTION:

- Restoration of T-cell and B-cell immunity, which generally takes 12 months or more, is essential to the recipient's recovery process.[58] It is only when the donor's immune system is fully functional within the recipient that the risk of opportunistic infection declines to premorbid levels. However, presence of immunocompetent donor T cells can lead to the recognition of host tissue as foreign and thus the development of GVHD.[59] GVHD is classified as acute when it occurs in first 100 days post transplantation and chronic when it either persists or develops after day +100.Clinically significant acute GVHD occurs in about 40% of HLA matched-sibling and 80% of matched unrelated-donor (MUD) transplant recipients.

RISKS

- Treatment-related mortality occurring in first 12 months after matched stem cell transplantation is about 20% to 30%.25 This figure is higher among recipients of an allograft from MUD, reaching about 50%.25 Patients who do not develop transplant-related complications may still have a recurrence of underlying malignant disease in the first 2 years after transplant. The risk of relapse depends on status of disease at the time of HSCT, but even a case of acute leukemia in first complete remission recurrence is observed in 25% of patients.[60]

BLOOD PRODUCT SUPPORT —

Blood product support is usually required before, during, and following HCT [61]. In addition, blood product support, with resulting iron overload, is commonly seen in those patients undergoing HCT for a hematologic disorder. Such iron overload (eg, serum ferritin >1000 ng/mL) may affect overall survival post-HSCT, increasing the likelihood of acute GVHD, as well as incidence of blood stream infections and SOS [62-64].

Virtually all patients require blood product support in the form of red blood cell and platelet transfusions until engraftment to support hematopoiesis. This generally requires 2-3 weeks post transplant.Transfusional needs are significantly lower in nonmyeloablative preparative regimens. [65].

Red blood cell transfusion — The indications for transfusion of blood products vary from center , many centers use a threshold of 7 to 8 g/dL of hemoglobin.

Patients with GVHD on drugs such as cyclosporine may have continued blood product requirements from bleeding and/or microangiopathic hemolysis.

Cases of delayed hemolysis and/or pure red cell aplasia have been reported in patients with pre-existing alloantibodies against Rh or ABO antigens.

Platelet transfusion — Patients who are thrombocytopenic with bleeding require platelet transfusions. In comparison, the role of prophylactic platelet transfusions remains controversial. Many transplant centers have relied upon threshold values of platelet numbers to determine the timing of platelet transfusions.

Granulocyte transfusions — Granulocyte transfusions have generally not been used because of the lack of efficacy in clinical trials and the difficulty of obtaining sufficient numbers of cells due to the short half-life of neutrophils.

Growth factor support (GM-CSF and G-CSF)— American Society of Clinical Oncology (ASCO) [66]. Supported the use of colony-stimulating factors for mobilization of PBPCs for transplantation. In the post-transplant setting the usual dose is 5 mcg/kg per day for G-CSF (filgrastim), or 250 mcg/m^2 per day for GM-CSF (sargramostim) [14]. Therapy is usually begun 1 to 5 days after transplantation and continued until ANC of 10,000/microL.

INFECTIONS IN HSCT-

The types of infections to which these hosts are most vulnerable can be be divided based upon three periods of interest are:

- Preengraftment — <3 weeks
- Immediate postengraftment — 3weeks to months
- Late postengraftment — >3 months

PREENGRAFTMENT — The major risk factors for infection during the preengraftment period in the first three weeks after HCT are mucositis and cutaneous damage, neutropenia with resulting loss of phagocytic abilities, and organ dysfunction [67].

Bacterial infections — Aerobic gram-positive and gram-negative bacteria account for most documented infections during this granulocytopenic period [68]. Gram-positive organisms include coagulase-negative staphylococci, Staphylococcus aureus, viridians, streptococci, and others [69]. Important risk factors for bacterial infections are neutropenia and

mucocutaneous damage [67]. A rapidly fatal streptococcal shock syndrome can occur in a small subset of patients with infection by viridans streptococci [70-72].

Diarrhea commonly occurs among patients who have undergone HCT due to both infectious and noninfectious agents. The most common cause of infectious diarrhea in patients undergoing HCT is Clostridium difficile-associated diarrhea. The frequency of C. difficile associated diarrhea is illustrated by a study of 135 HSCT patients who developed diarrhea either before or after engraftment and were tested for C. difficile toxin A, 21 (16 percent) were positive [74].

Gram-negative infections may be caused by Legionella spp, Pseudomonas aeruginosa, Enterobacteriaceae, , and other bacteria [75]. The most common sites of bacterial infection include those involving the bloodstream and lungs, eg, bacteremia and pneumonia

Fungal infections — Allogeneic transplant recipients are at a significantly higher risk for fungal infection than those receiving autologous marrow stem cells.

Candida — The introduction of antifungal prophylaxis with triazole antimicrobials, especially fluconazole, has significantly reduced the morbidity and mortality of invasive candidiasis [76, 77].

Risk factors for invasive candidiasis include severe neutropenia, use of broad-spectrum antibiotics, organ dysfunction, mucocutaneous damage, and yeast colonization with Candida spp [78-81].

Viral infections — the major viruses encountered during the immediate posttransplant period are herpes simplex virus (HSV) and RSV.

Herpes simplex virus — almost all HSV infections in HSCT recipients are caused by viral reactivation; thus, only seropositive patients are at risk. The rate of reactivation is more than 70 percent [82]. The median time to onset of HSV disease is 2-3 weeks [83].

HSV-1 infections primarily present as severe mucositis and occasionally esophagitis.Reactivation of HSV-2 infection in the genital or perineal area accounts for 10 to 15 percent of all HSV infections in HSCT patients [84]. Prophylaxis with acyclovir has markedly reduced the incidence of all herpetic infections [85].

Respiratory viruses — the most common respiratory viruses include respiratory syncytial virus (RSV), the parainfluenza viruses, rhinoviruses, and influenza A and B. The frequency and timing of the isolation of these viruses generally reflects the pattern found concomitantly in the community and often varies from year to year [86-88].

IMMEDIATE POSTENGRAFTMENT —The major risk factors for infection during the immediate postengraftment period three weeks to three months after HCT are mucositis and cutaneous damage, similar to preengraftment, but also cellular immune dysfunction, immunomodulating viruses, hyposplenism, decrease in opsonization, diminished reticuloendothelial function and acute graft versus host disease (GVHD) and its therapy

Bacterial infections — Bacterial pathogens deserving special attention during this period are Listeria monocytogenes [89] and Legionella pneumophila [90].

Fungal infections — Invasive aspergillosis can occur among both allogeneic and autologous HCT recipients although the incidence is more frequent among the former (5 to 30 versus 1 to 5 percent) [91-95]. The median time of onset of aspergillosis is 100 days after transplantation [96]. Risk factors for aspergillosis include older age, the presence and severity of GVHD, corticosteroid therapy, graft failure, diagnosis other than chronic myeloid leukemia, advanced cancer at transplantation, cytopenias (neutropenia, lymphopenia, monocytopenia) and iron overload [97,98]. Risk factors associated with early invasive mold infections following allogeneic stem cell transplantation (<40 days) include older age and human leukocyte antigen match, whereas variables associated with late disease (≥40 days following HSCT) include GVHD and CMVinfection [98].

Chronic disseminated candidiasis is now rarely seen in HSCT recipients since the introduction of azole prophylaxis. The development of fever, abdominal symptoms, and increasing alkaline phosphatase in a patient who has recently recovered from neutropenia and had not received appropriate antifungal prophylaxis should prompt an investigation for hepatosplenic candidiasis.

Pneumocystis carinii (jirovecii) pneumonia — The median time to onset of PCP is nine weeks after HCT. However, this pathogen now accounts for < 1 to 2 % of pneumonias in transplant recipients with routine use of effective chemoprophylaxis [99].

Cytomegalovirus — Before the routine use of prophylactic regimens, CMV seropositive allogeneic HCT recipients had a 70 to 80 percent risk of reactivation of this virus, and one-third of these patients developed CMV disease [100]; CMV disease, mainly pneumonia, evolved in < 5% of patients [101-103]. The risk of acquiring CMV from either blood transfusion or seropositive marrow in CMV seronegative HSCT recipients is approximately 40 percent [104-105].Risk factors for symptomatic CMV disease include CMV seropositive recipient, high titer of virus, allogeneic HCT especially from a matched unrelated donor (MUD), advanced age, use of total body irradiation for conditioning, acute GVHD, and the use of CD34+ selected allogeneic HCT [106-110]. There does not appear to be a difference between the incidence of CMV disease after allogeneic bone marrow transplantation compared with allogeneic peripheral blood transplantation.

CMV infections in HCT recipients most commonly present as fever of unknown origin, interstitial pneumonitis, or enteritis [110-116]. Preemptive antiviral therapy has markedly reduced the incidence and severity of CMV disease [117,118] and delayed its onset from a median of eight weeks to greater than three monthsy [114,115].

Epstein-Barr virus — Primary EBV infection presenting with pneumonia was reported in a HCT recipient one month following transplantation [116]. The virus must have been transmitted by the donor's bone marrow as the transplant recipient had negative serologic tests for EBV before transplantation and the donor's bone marrow was positive for EBV.

Parasitic infections — The occurrence of parasitic infections after HCT frequently requires unique exposures, with the exception of toxoplasmosis.

Toxoplasmosis — Reactivation of toxoplasmosis occurs in 5 to 15 percent of T cell depleted or otherwise severely immunosuppressed allogeneic transplant recipients; in other transplant recipients the incidence is less then 1 percent. The infection typically develops in the second month after transplantation [117,118], among patients seropositive prior to HCT. Patients with reactivation toxoplasmosis commonly have neurologic deficits and/or seizures, although disseminated disease is also frequent [119-121].

Mycobacterial infections — Mycobacterial infections in transplant recipients are rare, occurring in one to three percent of allogeneic and 0.2 percent of autologous HCT recipients

[122-124]. Infection can arise due to reactivation (Mycobacterium tuberculosis and Mycobacterium avium complex) or new exposure (atypical mycobacteria).

The most frequent manifestation of tuberculosis is pulmonary infection, which tends to occur during the first three months. Extrapulmonary disease, such as bloodstream, catheter-related, soft tissue, bone and joint infections, is more common with atypical mycobacteria [122-124].

LATE POSTENGRAFTMENT — Late infectious complications are typically only seen among allogeneic recipients.

Bacterial infections — Late bacteremia is not uncommon after allogeneic HCT and is typically caused by the encapsulated bacteria (Streptococcus pneumoniae, Haemophilus influenzae, Neisseria meningitidis) [123-125], staphylococci and gram-negative bacteria, such as Pseudomonas spp [126]. Pneumonia and meningitis are among the complications [127].

The risk of pneumococcal infection is greater among patients with severe chronic GVHD [127-129], immunoglobulin deficiency (usually in subclasses IgG2 and IgG4) [130], and hyposplenism [131,132] Bacteremic pneumonia was the most common manifestation, but isolated pneumonia and bacteremia also occurred.

Viral infections — Varicella zoster virus (VZV), EBV, and viruses to which the transplant recipient may have lost immunity figure more prominently as infections during this late postengraftment period.

Varicella zoster virus — The incidence of VZV reactivation is approximately equal among allogeneic and autologous HCT recipients (20 to 40 percent). VZV infection tends be more common among children (up to 90 percent by year one) and to occur earlier posttransplantation (median 100 days) [133]. Infection typically occurs during the first six to nine months (80 percent during first year) and may be associated with complications including:

- Cutaneous dissemination — 25 percent
- Post-herpetic neuralgia — 25 percent
- Scarring — 20 percent

- Bacterial superinfection — 15 percent
- Death — 5 percent
- CNS manifestations — <2 percent

Acyclovir therapy may reduce the risk of VZV infection after allogeneic HCT. In a randomized, placebo-controlled trial of 77 HCT recipients, acyclovir (800 mg twice daily) from one to two months after HCT until one year was associated with a significant reduction in VZV infection at one year (5 versus 26 percent, hazard ratio 0.16, 95% CI 0.04-0.74) [134].

Acute management of the febrile stem cell transplant recipients — All febrile HCT recipients should be treated empirically with broad-spectrum antibiotics. The choice of empiric therapy should depend upon the risk group (high or low risk for complications), the potential sites of infection, the susceptibility patterns at a given institution, and the cost of the various regimens. The approach can be based upon the risk for complications in a given patient.

GVHD

Graft-versus-host disease (GVHD) occurs when immune cells transplanted from a non-identical donor (the graft) recognize the transplant recipient (the host) as foreign, thereby initiating an immune reaction that causes disease in the transplant recipient.

GVHD has been classically divided:

- Acute GVHD — within the first 100 days of (HSCT)
- Chronic GVHD —after the first 100 days

Classic rash, abdominal cramps with diarrhea, and a rising serum bilirubin concentration within the first 100 days following transplantation is diagnostic of GVHD

Renal disease — with acute GVHD, the induction of autoimmune disease occurring in association with autoantibody production may require the expression of particular class II haplotypes. In a murine model of GVHD, for example, the onset of lupus-like nephritis in animals producing pathogenic IgG antinuclear antibodies was dependent upon the MHC haplotype expressed by the recipients [135]

Table 7: GVHD grading

SKIN	+1	Maculopapular rash over <25 percent of body area
	+2	Maculopapular rash over 25 to 50 percent of body area
	+3	Generalized erythroderma
	+4	Generalized erythroderma with bullous formation and often with desquamation
LIVER	+1	Bilirubin 2.0 to 3.0 mg/dL; SGOT 150 to 750 IU
	+2	Bilirubin 3.1 to 6.0 mg/dL
	+3	Bilirubin 6.1 to 15.0 mg/dL
	+4	Bilirubin >15.0 mg/dL
GUT	1	Diarrhea >30 mL/kg or >500 mL/day
	+2	Diarrhea >60 mL/kg or >1000 mL/day
	+3	Diarrhea >90 mL/kg or >1500 mL/day
	+4	Diarrhea >90 mL/kg or >2000 mL/day; or severe abdominal pain with or without ileus

THE HOSPITAL STAY AND BEYOND

The usual duration of hospitalisation for allogenic transplantation is about 4 weeks, but if any of the complications arises the stay is prolonged. After discharge, patient is followed up to assess graft function, GVHD, transfusion support, late regimen- related toxicity and administration of prophylactic antimicrobials.

ACUTE KIDNEY INJURY IN ALLOGENIC HAEMATOPOETIC STEM CELL TRANSPLANT

INTRODUCTION —

Hematopoietic stem cell transplantation (HSCT) is the only definite treatment for many of the hematologic and oncologic diseases but, it is associated with risk of both AKI and CKD [136].

AKI as defined by two fold increase in the serum creatinine is common, and a incidence of 50 to 60 % is reported. In most of patients, renal dysfunction is temporary and returns to normal.

EPIDEMIOLOGY AND PROGNOSIS —

The incidence and risk factors of AKI vary with conditioning regimens:

Ex: Myeloablative allogenic HSCT requires an high dose conditioning regimen, frequently in combination with TBI and it is associated very risk of AKI compared to nonmyeloablative allogenic HSCT.

Epidemiology of acute kidney injury — Most cases of acute kidney injury following HSCT develop +10 to +21 days after HSCT. By day +21, as many as 50 percent of patients have a doubling of the serum creatinine concentration [137-141]. However, the incidence of AKI following HSCT may be decreasing. In a retrospective review of over 2500 HSCT recipients, the incidence of a two fold rise serum creatinine was significantly lower during the period of 2003 through 2007 when compared to 1993 through 1997 (33 versus 50 percent) [141].

Type of regimen — The risk of AKI associated with HCT depends in part upon the specific procedure used, with myeloablative regimens associated with a higher incidence of acute kidney injury compared with nonmyeloablative regimens [142,143], and allogeneic regimens

associated with a higher risk than autologous regimens. In a review of these and other studies, the average incidence rates of severe acute kidney injury, defined as a 50 percent or greater reduction in glomerular filtration rate and/or more than doubling of the serum creatinine and/or the requirement for dialysis, were as follows:

- 76 percent with myeloablative allogeneic HCT
- 43 percent with nonmyeloablative allogeneic HCT
- 23 percent with autologous HCT

The incidence of AKI is higher with myeloablative compared with nonmyeloablative allogeneic HCT despite the fact that patients who undergo nonmyeloablative therapy are older and have more comorbidity. In one study, the risk of acute kidney injury with a myeloablative regimen was nearly fivefold higher than the risk with a nonmyeloablative regimen after accounting for age and other comorbid conditions [142].

Prognosis — Renal failure requiring dialysis in the peritransplant period has a poor prognosis due, in part, to its association with coexistent injury of multiple other organs [142-144]. In one study of 88 patients who underwent allogeneic HSCT, the requirement for dialysis was associated with 83 percent mortality, with patients dying an average of 47 days post-transplant.

A 2005 meta-analysis of six published reports of myeloablative allogeneic transplantation noted a twofold increased risk of death with AKI (defined as doubling of the serum creatinine concentration) and a sevenfold increased risk of death with acute kidney injury requiring dialysis [9]. Mortality rates associated with AKI vary by modality and have been reported to be 54, 48, and 11 percent at 1000 days after autologous, myeloablative allogeneic, and non-myeloablative allogeneic HSCT, respectively [45].

Epidemiology of chronic kidney disease — there is a wide range in the reported incidence of chronic kidney disease (CKD) following HSCT (zero to over 60 percent at ≥6 months following HSCT); this is related in part to variations in the definition of CKD, duration of follow-up, and type of HSCT [48].

Radiation therapy has been implicated, possibly by causing a subacute or chronic thrombotic microangiopathy (radiation nephritis) [149-154]. The likelihood of developing CKD varies with the radiation dose [155]. This was suggested in a review of 11 studies in which CKD was rare when the total radiation dose to the kidney was less than 17 Gy; most patients who developed CKD received more than 20 Gy.

The long-term renal prognosis following total body irradiation (TBI) appears to be relatively benign [153,154]. This was suggested in a report of 60 patients who underwent HSCT in which 34 (57 percent) had at least a 20 percent fall in GFR at a mean follow-up of two years; this was mostly seen in patients who underwent TBI [18]. However, the mean glomerular filtration rate after TBI was still above 80 mL/min (compared with 100 mL/min among patients who did not receive TBI).

A similar benign outcome was noted in a review of patients receiving TBI (estimated renal dose 12 Gy) in which only two of 33 surviving patients had persistent elevations in serum creatinine 24 months after HCT [154].

Factors other than TBI can contribute to the development of CKD. This was illustrated in a series of 122 patients who underwent nonmyeloablative HSCT (with only 2 Gy of radiation given); 81 patients (66 percent) had a ≥25 percent reduction in glomerular filtration rate at six months [156]. Metanalysis found that acute kidney injury, previous autologous HSCT, long-term CNI use, and chronic GVHD were independently associated with the development of CKD.

ETIOLOGY OF ACUTE KIDNEY INJURY —

The most common causes of acute kidney injury after HSCT are acute tubular necrosis, toxicity from medications such as calcineurin inhibitors, and hepatic sinusoidal obstructive syndrome (SOS) [157]. Acute tubular necrosis is typically seen during the period of pancytopenia in septic patients who may also be treated with one or more nephrotoxins, including amphotericin B (both liposomal and conventional), aminoglycosides, and acyclovir. Less frequent etiologies include tumor lysis syndrome, thrombotic microangiopathy, graft versus host disease (GVHD), and hemolysis associated with ABO incompatible transplants

[158]. In addition, acute kidney injury after HSCT may result from the overlapping occurrence of more than one of these etiologies.

Hepatic sinusoidal obstruction syndrome (veno-occlusive disease) — A hepatorenal-like syndrome due to hepatic sinusoidal obstruction syndrome (SOS) is an frequent cause of AKI [159,160]. Hepatic SOS is exclusively of HSCT.

The renal failure with hepatic SOS usually begins after the onset of hepatic disease (as manifested by progressive hyperbilirubinemia) and a superimposed event, such as sepsis triggers the onset of AKI. Patients with a plasma bilirubin concentration >7 mg/dL (120 µmol/L) are at higher risk for the subsequent need for hemodialysis.

Pathogenesis of liver disease causing renal dysfunction is not clear, but decreased hepatic clearance of endotoxin absorbed from the gut may play an important role [24]. It is possible that chemoradiation induced injury to sinusoidal epithelial cell leads to the development of portal hypertension, splanchnic vascular dilatation, sodium and water retention and systemic vasoconstriction [159], mimicking hepatorenal syndrome. In some patients, however, renal dysfunction is evident before the onset of overt liver disease.

The fractional excretion of sodium typically remains below 1 percent in these patients, similar to the findings seen in the hepatorenal syndrome. The urinalysis often reveals many muddy brown granular casts; however, this abnormality appears to be induced by bilirubin toxicity rather than reflecting tubular necrosis.

ABO incompatible transplants — Patients who receive hematopoietic cells from a donor with a major ABO incompatibility are at risk for hemolysis and acute kidney injury due to hemoglobinuria [161]. Nevertheless, these types of transplants are generally safe if careful attention is paid to minimizing the risk of hemolysis. This includes red blood cell depletion of the graft, vigorous hydration, and infusion of mannitol if overt hemolysis does occur.

ETIOLOGY OF SUBACUTE OR CHRONIC KIDNEY DISEASE

Thrombotic microangiopathy (TMA) — this because of combination of factors that cause damage to endothelium, including CNI, chemotherapy, GVHD, and TBI [158,162]. The classic picture of TTP-HUS induced by calcineurin inhibitors is a rare complication to occur soon after HSCT [163].

Clinical presentation is a subacute or chronic TMA that first becomes apparent +20 and +100 days after HSCT is more common [164-169]. Histological examination of renal biopsy can reveal mesangiolysis with necrotizing arteriolar and glomerular lesions, and intraglomerular and renal arteriolar thrombi [164-169]. The kidney is reported to be the primary site of microangiopathy, since autopsy findings have not identified systemic thrombi [169,170].

The renal abnormalities are thought to reflect mesangial and endothelial damage induced by chemotherapy and TBI that are given to eradicate the patient's underlying disease and provide immunosuppression prior to HSCT. In one study of 22 patients diagnosed with a TTP-like syndrome following HSCT, all had been treated with CNI and 20 had received TBI [171]

The importance of radiation (as in radiation nephritis) is suggested by two findings:

- Partial shielding of the kidneys during total body irradiation can diminish the incidence of late renal dysfunction [172,173].
- A late decline in renal function is seen primarily in patients treated with total body irradiation, not those treated only with chemotherapy.

However, the observations that some affected patients were not given radiation, and that high-dose cyclophosphamide can induce mesangiolysis in animal models, suggests that an alkylating agent may also be pathogenetically important [174]. The lack of cyclophosphamide nephrotoxicity in other settings is a probable reflection of the much lower dose (one-tenth) used to treat glomerulonephritis and vasculitis.

It is possible that infection could be a initiating or contributing factor. In a review of 35 autopsies of patients with TMA associated with HSCT, infection was listed as the cause of death in 54 percent, with the most common organisms being Aspergillus species and CMV. This observation is consistent with the ability of these organisms to cause microangiopathic hemolytic anemia [175,176].

The incidence of thrombotic microangiopathy has varied in different studies (zero to 75 percent) [165,171]. The diagnosis can be difficult to make as the presenting signs and laboratory findings, such as anemia, thrombocytopenia, and acute kidney injury, are common in patients who undergo HCT. Autopsy studies have noted a poor correlation between clinical criteria and histological findings [169,170].In most patients, the hematologic abnormalities eventually resolve, although hypertension and stable CKD (in which the plasma creatinine concentration is usually below 2.5 mg/dL or 220 µmol/L) usually persist [166]. Some patients may progress to end-stage renal disease (ESRD) [179].

Treatment and prevention — there are no convincing data to support the efficacy of plasma exchange in patients who develop TMA after HSCT, in contrast to the beneficial response seen in most other forms of TTP-HUS [164,165]. Plasma exchange has been tried in selected patients with disappointing results [169,171,180,181]. The lack of efficacy of plasma exchange may be due to the fact that TTP-HUS following HSCT is due to direct injury to the kidney from drugs or radiation, and deficiency of the ADAMTS-13 protease, which is present in classic TTP, is not involved in the pathogenesis of thrombotic microangiopathy after HSCT. Since TMA is often associated with CNI toxicity hence the discontinuation of cyclosporine or tacrolimus is essential.

In a survey of the literature, no reported patient convincingly responded to plasma infusion or exchange and patients were not severely deficient in the vWF cleaving protease, indicating that these patients do not have classic TTP. In addition, the microangiopathy appears to primarily limited to the kidney, since systemic thrombotic microangiopathy was

not identified at autopsy in the review cited above [169]. Instead, supportive care and withdrawal of calcineurin inhibitors, such as cyclosporine A, are the mainstays of therapy. Small studies utilizing daclizumab [182] and Rituximab [183] for treatment have met with moderate success.

Given the current lack of effective therapy, prevention is the major therapeutic goal in this disorder. Studies suggest that the risk of this condition may be minimized by shielding the kidneys [172], by fractionating the total body irradiation over several days and by replacing cyclophosphamide with other types of chemotherapeutic drugs. It may also be desirable to avoid the use of other nephrotoxins such as platinum in close proximity to the conditioning regimen [184]. Vigorous hydration is also likely important.

Studies in animal models of radiation nephritis and bone marrow transplantation nephropathy suggest that angiotensin converting enzyme (ACE) inhibitors may be effective for both prevention and treatment [185-187]. The applicability of these findings to humans is uncertain, but it seems prudent to preferentially use an ACE inhibitor in patients who become hypertensive. Prophylactic therapy is not recommended [188].

Calcineurin inhibitor toxicity (CNI) —CNI therapy (cyclosporine and tacrolimus)can cause nephrotoxicity similar to that observed in other settings, such as solid organ transplantation. The early renal vasoconstriction induced by these agents is markedly enhanced by concurrent therapy with amphotericin B, and a temporary reduction in dose or cessation of therapy may be indicated if acute kidney injury occurs. Frequent monitoring of the plasma creatinine concentration with dose adjustments for rising creatinine values is also helpful.

Long-term complications are uncommon, as CNI is given in full doses to stable recipients for only few months. However, the characteristic vascular and interstitial changes of cyclosporine nephrotoxicity can happen with prolonged therapy for GVHD [189].

Nephrotic syndrome — there are case reports and case series of nephrotic syndrome following HCT [190-196]. As an example, in one series of 163 patients who underwent HCT, nephrotic syndrome developed in seven (4.3 percent) a median of 300 days after HCT [191].

A variety of histologic patterns have been reported; the most common are membranous nephropathy (MN) followed by minimal change disease (MCD) [191,192,197]. This was illustrated in a literature review of 42 well-documented cases; 60 percent had MN and 22 percent had MCD [192].

The occurrence of MN is often associated with a decrease in immunosuppression and, therefore, with an increased likelihood of GVHD. A proposed mechanism is immune dysregulation from transfer of alloreactive donor lymphocytes with reactivity toward glomerular antigens [193]. In the above literature review, nephrotic syndrome was diagnosed 8 to 14 months following HCT, and all 42 cases occurred within two months following the diagnosis of GVHD [192].

Remission frequently occurs upon re-initiation of immunosuppressive drugs (corticosteroids, in particular) or anti-B cell therapy (anti-CD20, rituximab) [193], but some do progress to ESRD [191]. The treatment is the same as for idiopathic MN, but success is variable.

MANAGEMENT OF PATIENTS ON DIALYSIS —

The prognosis in patients who develop dialysis-requiring kidney disease varies with the time of onset. The requirement for dialysis for acute kidney injury occurring in the peritransplant period is an ominous event since it typically occurs in association with injury to multiple other organs. Relatively few of these patients are long-term survivors.

Patients who require dialysis at a later stage (ie, ESRD), when they are hematologically stable should do better. Among patients with ESRD, using a kidney for renal transplantation from the original hematopoietic cell donor may obviate the need for anti-rejection therapy, since the donor cells have repopulated the patient's bone marrow [199,200]. By comparison, with an allograft that is foreign to donated marrow cells, antirejection therapy is still required, but perhaps at lower doses since reconstituted marrow may not be fully

functional. Combined hematopoietic cell and kidney transplant has also resulted in the development of tolerance to the transplanted kidney and withdrawal of immunosuppressive medications [200].

AIM

To identify the incidence and outcomes of acute kidney injury classified by Acute Kidney Injury Network criteria (AKIN) associated with allogenic hematopoietic stem cell transplantation (HSCT).

MATERIALS AND METHODS

SETTING:

Setting and location: Department of Nephrology and Haematology

Christian Medical College, Vellore.

PERIOD OF RECRUITMENT & EXPOSURE

All Patients underwent HSCT between January 2010 and December 2011.

FOLLOW-UP

The patients were followed up for a minimum of 1 year from the date of HSCT till their last visit on before December 2012. Clinical and laboratorial data at each of their visit was collected. For the study, no additional history questionnaire and investigation was used apart from the routine evaluation.

Investigations during follow up are performed as per treating clinician's advice and no extra blood investigations were performed in these patients. Patients' personal information such as name, hospital No., address, occupation etc., were not collected or analyzed. The data was de-identified and referred to using unique case I.D. numbers. Details of treatment before HSCT was noted and the treatment started after HSCT by the treating physicians was also noted

PARTICIPANTS:

Eligibility criteria

Inclusion criteria:

All subjects who underwent HSCT from Jan 2009 to Dec 2011 at Dept of Haematology, CMC Vellore**.**

Exclusion criteria:

Patients who underwent stem cell transplant for second time.

Figure 1: Protocol for HSCT

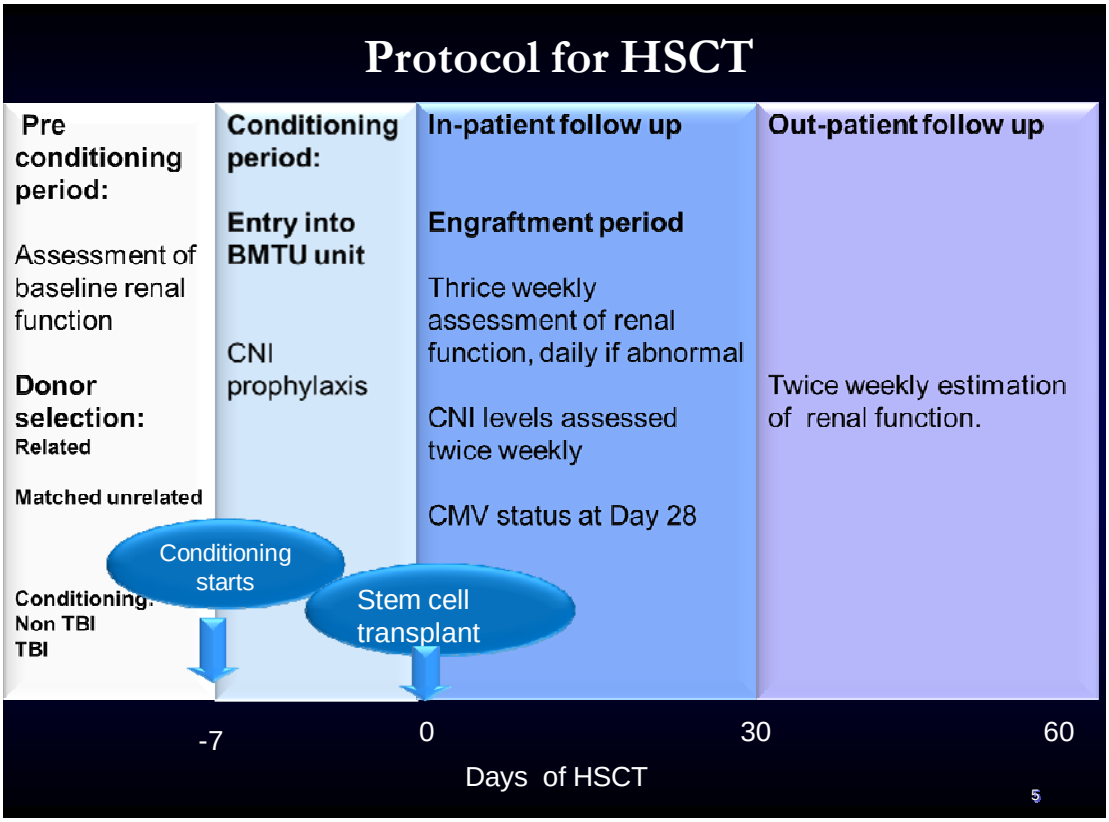


Figure 1: Protocol for HSCT

VARIABLES:

It is an observational study

DEFINITIONS: The following standard definitions and diagnostic criteria were used,

1. Glomerular filtration rate: Glomerular filtration rate (GFR) is the volume of fluid filtered from the renal (kidney) glomerular capillaries into the Bowman's capsule per unit time [201].

Adults:

Equation for estimating glomerular filtration rate (GFR) from serum creatinine is Modification of Diet in Renal Disease (MDRD) Study equation [202]

GFR (mL/min/1.73 m^2) = 175 × $(S_{cr})^{-1.154}$ × $(Age)^{-0.203}$ × (0.742 if female) × (1.212 if African American) (conventional units)

In Children:

GFR (mL/min/1.73 m^2) = (0.41 × Height in cm) / Creatinine in mg/dL

Currently the best equation for estimating glomerular filtration rate (GFR) from serum creatinine in children is the Bedside Schwartz equation [203]

2. Acute Kidney injury: Classification and definitions as described in the present knowledge section (12)

Risk factors of AKI:

Clinical predictors:

1) Age

2) Duration of illness

3) Age at diagnosis of Haematological disease

4) Hypertension

5) Renal function at the time of diagnosis

6) Proteinuria at the time of diagnosis

7) Hematuria – gross or microscopic

8) Prior use of antihypertensive drugs

10) Other co morbidities like Diabetes mellitus, coronary heart disease etc.

11) **Febrile neutropenia**: Absolute neutrophil count, ANC< 500/cu.mm with fever (Temp 38.5°C).

12) **Cell dose:** Amount of Mononuclear cells/CD34 hematopoietic cells used for HSCT calculated by flowcytometry.

13) **BK Virus viruria:** Urine BK virus Qualitative polymerized chain reaction positive.

14) **Cytomegalovirus viremia**: Blood Quantitative polymerized chain reaction >1000 CMV genome copies / ml.

15) **Sinusoidal-occlusive syndrome of the liver (SOS)**, previously known as Veno-occlusive disease is a hepatotoxic lesion causing the obstruction of small intrahepatic venules, hence leading to lesions in the central zone hepatocytes and surrounding sinusoids.

Table 8*: *Modified Seattle criteria for diagnosis of SOS
Occurrence of two of the following events within 20 days of HSCT (i) Hyperbilirubibemia (total S. bilirubin >2 mg/dl) (ii) Hepatomegaly or upper quadrant pain of liver origin (iii) Unexplained weight gain (> 2% of baseline body weight)

16) **Mucositis**: Mucositis is inflammation of the mucosal surfaces throughout the body. It typically involves redness and ulcerative sores in the soft tissues of the mucosa.

17) **Sepsis**: Systemic inflammatory response syndrome with pathogen obtained from blood culture.

18) **UTI:** Growth of more than 105 colony forming units of organisms with symptoms of lower urinary tract infections.

19) **Systemic Mycosis:** Culture positive fungal infection from blood /suspected tissue/radiological evidence with initiation of antifungals.

OUTCOME: The outcomes of the study were,

1. Recovery of renal function: defined as the decline in S. creatinine to the baseline ± 0.1mg/dl.

2. Persistent renal dysfunction: defined as persistent elevation of S.creatinine after an episode of AKI, which was considered to be irreversible loss of renal function to the extent that they may be labelled as chronic kidney disease.

3. Death

DATA SOURCES/MEASUREMENT: Data source is through the following,

1. Chart records:

History and treatment details (current and past i.e., outside), Blood pressure, height, weight, Body Mass Index (BMI), age, gender ,demographic details, induction regimen, HLA match, drugs used are noted.

2. Lab investigations from computerised hospital information processing system:

Serum creatinine, albumin, 24 hours urine protein or Urine protein/Urine creatinine ratio, liver function tests, infections, urine analysis and other investigations are noted.

GFR was calculated by four variable MDRD with use of serum creatinine from lab reports, age and gender from the chart records.

BIAS: Bias was minimised through,

1) Consecutive HSCT patients were recruited into the study

2) Data was collected in a de-identified manner and as indicated by unique case ID numbers without patient's name or hospital number.

STATISTICAL METHODS:

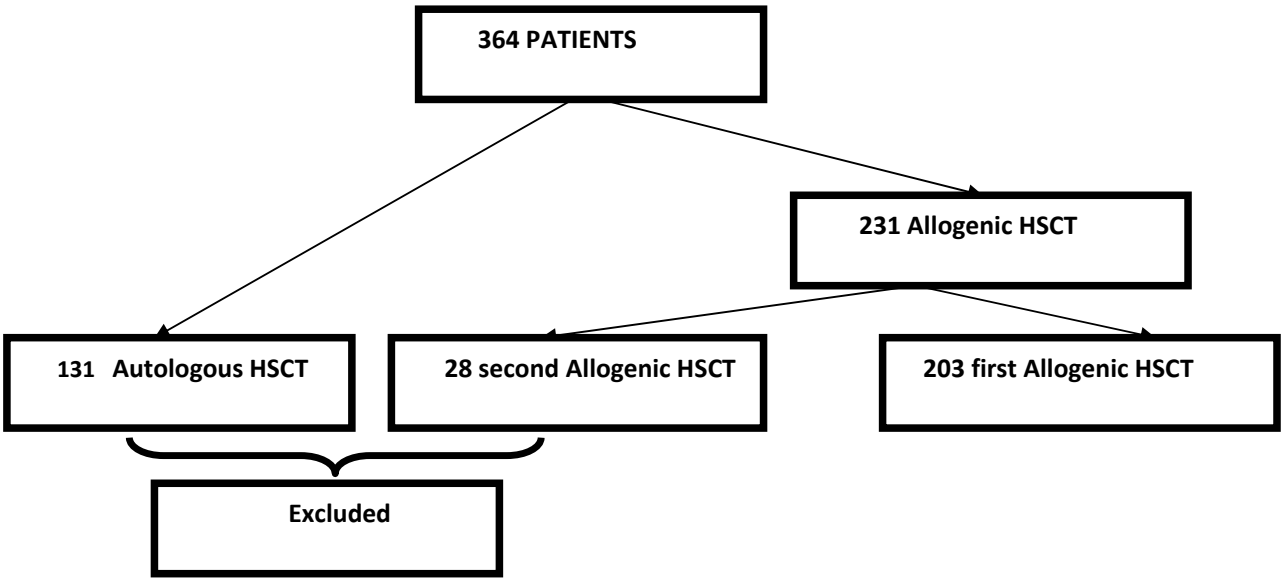
All variables were described using descriptive analysis. Categorical variables were expressed as n (%) with continuous variables as mean ± standard deviation or median (range) depending upon normality of distribution. The association of risk factors to AKI and outcome were assessed .Categorical variables were analyzed using Chi square test or Fischer's exact test and continuous variables were analyzed using independent sample t test. Multivariate analysis was performed for identifying independent risk factor for AKI and mortality**.**

RESULTS

PATIENTS:

This study was carried out between January 2010 to December 2012. During the study period a total of 231 patients underwent allogenic stem cell transplant, out of which 28 patients were excluded from the study for the homogeneity of study population.

Figure 2: Patients /study group



GEOGRAPHICAL DISTRIBUTION:

Majority of the patients were from India and South East Asian countries, with minority from Africa and Middle East.

Table 9: Geographical distribution

Region	N (%)
Northern India	49 (24.1)
Eastern India	09 (4.4)
Southern India	95 (46.8)
Western India	29 (14.3)
Foreign nations	21 (10.3)

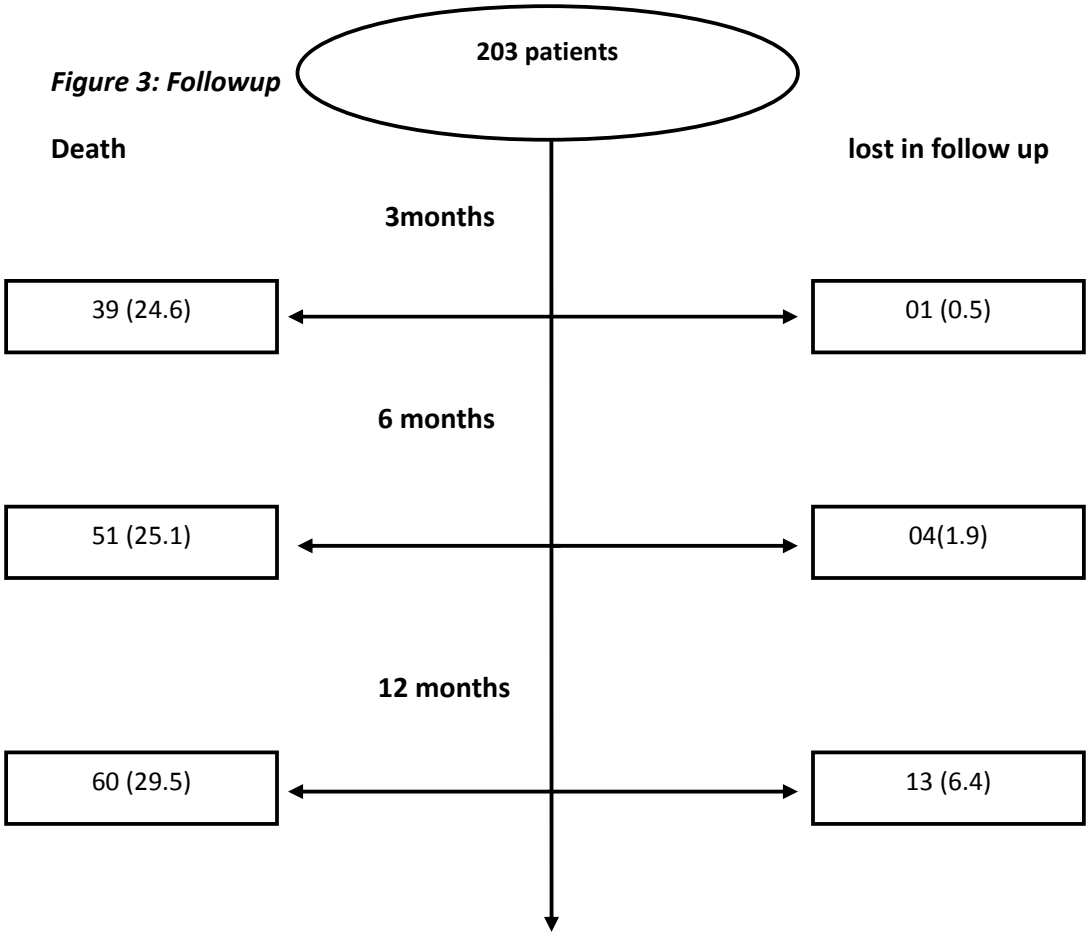


Figure 3: Followup

MAJOR INDICATIONS OF HSCT:

Overall the most common indication for HSCT was Thalassemia (%), among the haematological malignancies it is AML (%).A total of 9(4.5%) underwent transplantation for non haematological conditions.

Table 10: Overall indications of HSCT

Diseases	**N (%)**
Thalassemia major	59 (29.1)
Aplastic anemia	43 (21.2)
AML	36 (17.7)
ALL	26 (12.8)
CML	15 (7.4)

AML- acute myeloid leukemia,ALL-acute lymphoid leukemia,CML-Chronic myeloid leukemia

Haematological malignancies	**N (%)**	**Non Haematological malignancies**	**N (%)**
Aplastic anemia	43 (21.2)	Thalassemia major	59 (29.1)
AML	36 (17.7)	Fanconi Anemia	04 (2.0)
ALL	26 (12.8)	Wiskott Aldrich syndrome	03 (1.5)
CML	15 (7.4)	Severe Combined Immunodeficiency	02 (1.0)

BASELINE CHARACTERISTICS

Baseline characteristics indicate the recipients were of young age with mean age being 22.0 ± 14.5 years, and no associated significant co morbidities, indicating that they were at low risk of developing AKI.

Table 11: Baseline characteristics

Baseline characteristics	**N (%) / mean± SD**
Mean age (yrs)	22.0 ± 14.5
Gender (M:F)	1.57
Diabetes Mellitus	3 (1.5)
Hypertension	3 (1.5)
Coronary artery disease	2 (1.0)
Baseline S.creatinine (mg/dl)	0.71 ± 0.28
Total body irradiation (TBI)	37 (18.2)
Matched Unrelated DONOR* (MUD)	31 (15.3)
Average cell dose (x 10^8 MNC/kg body weight)	6.7± 5.2

*NMDP (U.S), DKMS (Germany), DATRI (India)

POST HSCT COMPLICATIONS:

Infections:

As expected febrile neutropenia was observed in all cases but blood culture positive septicemia was seen in 40%.Among the viral infections CMV was commonest (39.4%) followed by BK viruria (4.4%).Systemic mycosis accounted for about 52% of cases.

Table 12: Infectious complications post HSCT

Infections	**N (%)**
Febrile neutropenia	202 (99.5)
Sepsis	81 (39.9)
CMV viremia	80 (39.4)
BKV viruria	09 (4.4)
UTI	11 (5.4)
Systemic mycosis(suspected)	106(52.2)

CMV-Cytomegalovirus, UTI-Urinary tract infections

Immune related complications:

GVHD was commonest noninfectitious complication observed, followed by mucositis

Table 13: Immune mediated complications post HSCT

Immune mediated complications	N (%)
Mucositis	61 (30)
GVHD	71 (35)
SOS	14 (6.9)

GVHD-Graft versus host disease, SOS-Sinusoidal occlusive disease

Exposure to nephrotoxic agents:

A large number of patients were treated with nephrotoxic agent's predominantly being CNI, used for GVHD prophylaxis.

Table 14: Nephrotoxic agents exposure post HSCT

Nephrotoxic agents	N (%)
CNI	193 (97.0)
Amikacin	170 (83.7)
Amphotericin	94 (46.3)
Colistin	45 (22.2)

CNI- Calcineurin inhibitor

ACUTE KIDNEY INJURY (AKI):

A total of 170 episodes of AKI occurred in 149 (72.9%) patients by AKIN criteria predominantly these being in stage 1 and 2.

A significant number of patients had more than 1 episode of AKI with average number of episodes per patient being 1.29.About 14(6.8%) had AKI even prior to transplantation during the conditioning period

RIFLE VERSUS AKIN CRITERIA:

As expected more patients were diagnosed to have AKI by AKIN criteria compared to RIFLE in stage 1, but on compassion there was a good correlation between the two.

Table 15: RIFLE versus AKIN criteria

	NO AKI	AKIN STG1	AKIN STG2	AKIN STG3	Total
NO AKI	26.6%	4.9%	0	0	31.5%
RISK	0	33.9%	0	0	33.9%
INJURY	0	0	24.1%	0	24.1%
FAILURE	0	0	0	10.3%	10.3%
Total	26.6%	38.8%	24.1%	10.3%	

AKIN-Acute Kidney Injury Network, RIFLE-Risk injury Failure Loss End stage renal disease

DAY OF ONSET OF AKI:

Median duration of onset of AKI was 17 days, severe degree of AKI (AKIN STAGE 3, RIFLE failure) tending to occur earlier in the post transplant period.

Table 16: Onset of AKI

AKI onset in days	Median (range in min, max)
Overall AKI	17 (2,290)
Stage 1 AKIN	20 (2,290)
Stage 2 AKIN	17 (3,163)
Stage 3 AKIN	14 (2,100)

AKIN-Acute Kidney Injury Network, AKI-Acute Kidney Injury

CAUSES OF AKI:

As in any AKI, predominantly the cause of acute kidney injury was multifactorial but the presumed primary causal factor is

Table 17: Predominant cause of AKI

Cause of AKI	N (%)
Sepsis	70 (34.5)
CNI	39 (18.2)
Nephrotoxic antibiotics	13 (6.4)
GVHD /AGE	06 (3.0)
Idiopathic	16(7.9)

CNI- Calcineurin inhibitor, GVHD-Graft versus host disease, AGE-Acute gastroenteritis

RISK FACTORS FOR AKI:

On univariate analysis, older age, matched unrelated donor (MUD) and total body irradiation were significant risk factors for AKI were but none of infectious or immune related complications were statistically significant**.**

Table 18: Risk factors for AKI:

	No AKI n=54	**AKI n=149**	**P**
Age	13.5 ± 12.2	25.08 ± 14.1	<0.001
Male recipient	28 (51.8)	96 (64.4)	0.104
Diabetes	0 (0)	3 (2.0)	0.695
Hypertension	1 (1.8)	02 (2.1)	0.607
Matched unrelated Tx	7 (12.9)	24 (16.1)	0.380
TBI conditioning	03 (5.5)	34 (22.8)	0.003
Cell dose (10^8 MNC/kg)	7.0 ± 2.7	6.5 ± 5.9	0.583

TBI-Total Body Irradiation

Table 19: Risk factors for AKI-Infections and immune mediated complications:

	No AKI n=54	**AKI n=149**	**P**
Sepsis	22 (40.7)	59 (39.6)	0.545
Systemic mycosis	20 (37.0)	85 (57.0)	0.001
CMV	16 (29.6)	64 (42.9)	0.086
BKV	1 (1.8)	8 (5.3)	0.450
UTI	4 (7.4)	7 (4.7)	0.457
Mucositis	14 (25.9)	47 (31.5)	0.440
GVHD	15 (27.7)	56 (37.5)	0.190
SOS	3 (5.5)	11 (7.4)	0.462

CMV-Cytomegalovirus, UTI-Urinary tract infection, GVHD-Graft versus host disease, SOS-Sinusoidal occlusive disease

Risk factors for AKI- drugs:

Among nephrotoxic drugs, Amphotericin use and Cyclosporine level at day 7 were significant risk factors

Table 20: Risk factors for AKI- drugs

	No AKI n=54	**AKI n=149**	**P**
Cyclosporine C_0 level ng/ml(Day7)	162 ± 89	223 ± 117	0.002
Amikacin	47(87.0)	123(79.8)	0.523
Amphotericin	16(29.6)	78(82.5)	0.004
Colistin	09 (16.6)	36(24.1)	0.256

INDEPENDENT RISK FACTORS FOR AKI:

On multivariate analysis older age, TBI and Amphotericin use were independent risk factors for AKI

Table 21: Independent risk factors for AKI

Feature	**P**	**Exp (B)**	**C.I .95%**
Age	0.000	1.16	1.08 - 1.25
CsA C_0 level day7	0.063	1.01	1.00 - 1.01
TBI	0.015	6.63	1.44- 30.41
Constant	0.582	1.16	
MLR done by enter method. Variable(s) entered on step 1: Age, Matched Unrelated Donor, Total body Irradiation, Cyclosporine level at Day 7, Amphotericin, sepsis, Sinusoidal Occlusive Syndrome,systemic mycosis.			

AKI AND MORTALITY:

Mortality was observed in 37.9% of patients (n=77/203) at the end of study period.

Table 22: Cumulative Mortality at 3rd ,6th and at 1 year from HSCT

Mortality	**N (%)**
At 3rd month from HSCT	39 (24.6)
At 6th month from HSCT	51 (25.1)
AT 12th month from HSCT	60 (29.5)

AKI was a significant risk factor for mortality; other factors which were significant were older age, MUD, blood culture positive sepsis, CMV viremia, BKV viruria and SOS.

Table 23: AKI and mortality

	N (%)	**N in each stage**
No AKI	11 (20.3)	N=54
Stage 1 AKI	22 (28.2)	N=78
Stage 2 AKI	21 (42.0)	N=50
Stage 3 AKI	16 (76.2)	N=21

Table 24: Univariate analysis of risk factors for mortality

	No mortality n=133	**Mortality n=70**	**p**
Age	19.5 ± 13.2	26.6 ± 15.8	0.001
Male recipient	79(59.3)	45(64.2)	0.546
Diabetes	2(1.5)	1(1.4)	0.726
Hypertension	2(1.5)	1(1.4)	0.726
CAD	0(0)	2 (2.8)	0.118
MUD	14(10.5)	17(24.2)	0.010
TBI conditioning	22(16.5)	15(21.4)	0.391
Cell dose (10^8MNC/kg)	6.5 ± 3.02	7.0 ± 8.0	0.574

CAD-coronary artery disease, MUD-Matched unrelated donor, TBI-Total Body Irradiation

Table 25: Risk factors for mortality- AKI/infections/immune mediated complications:

	No mortality n=133	**Mortality n=70**	**p**
Sepsis	42(31.5)	16(22.8)	0.003
Systemic mycosis	53 (39.8)	53(75.7)	0.000
CMV	44(33.0)	36(51.4)	0.009
BKV	2(1.5)	7(1.0)	0.009
UTI	5(3.7)	6(8.5)	0.124
Mucositis	37(27.8)	24(34.2)	0.340
GVHD	43(32.3)	28(40.0)	0.175
SOS	4(3.0)	10(14.2)	0.004
AKI	90(67.6)	59(84.2)	0.011

CMV-Cytomegalovirus, UTI-Urinary tract infection, GVHD-Graft versus host disease, SOS-Sinusoidal occlusive disease, AKI-Acute Kidney Injury

INDEPENDENT PREDICTORS OF MORTALITY:

SOS, AKI, Sepsis, Systemic mycosis were independent predictors of mortality on multivariate analysis

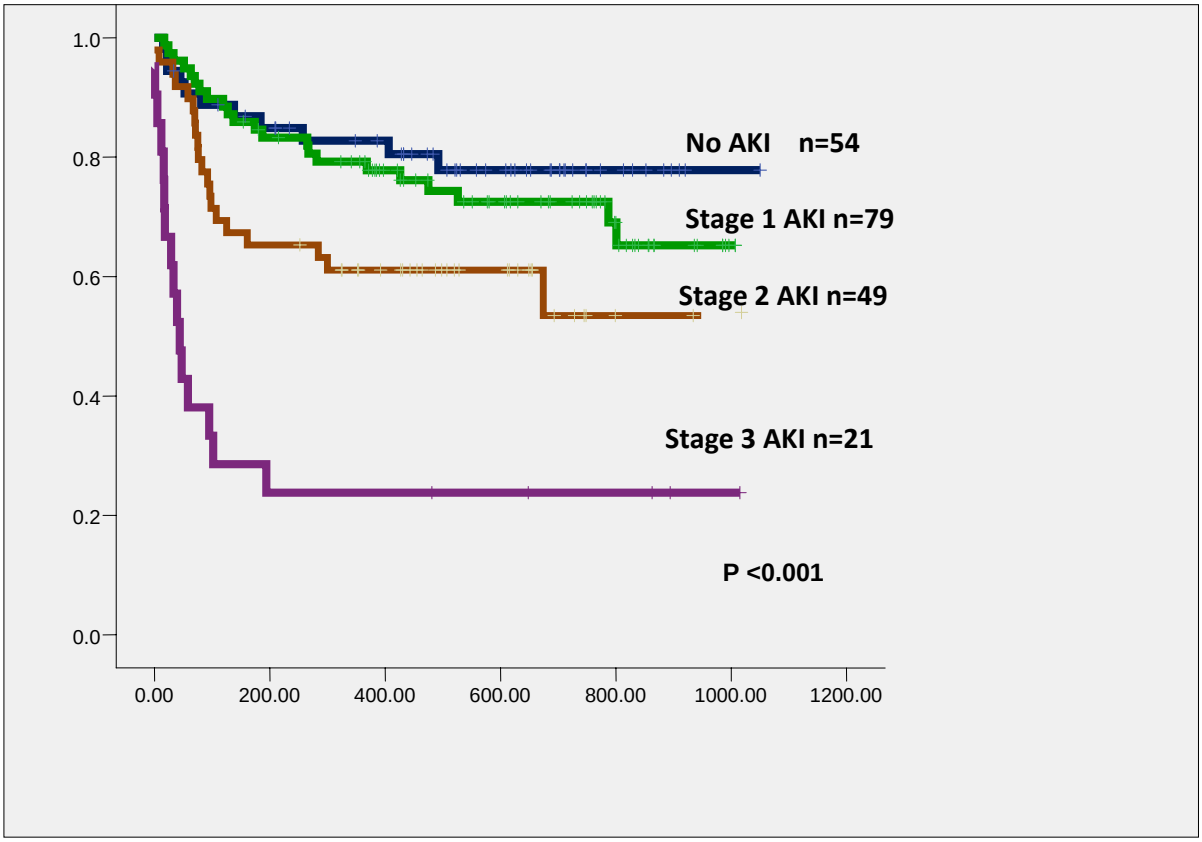
Table 26: Independent predictors of mortality

Feature	**p**	**Exp(B)**	**95% C.I**
SOS	0.014	6.39	1.46 - 27.89
AKI	0.086	2.22	0.894 - 5.55
Sepsis	0.055	2.08	0.983 – 4.42
Amphotericin	0.038	0.204	0.046 – 0.916
Systemic mycosis	0.002	12.5	2.59-60.43
Constant	0.000	0.062	
MLR done by Enter method. Variable(s) entered on step 1: Matched Unrelated Donor, Total body Irradiation, Cyclosporine level at Day 7, Amphotericin, sepsis, Sinusoidal Occlusive Syndrome, Acute Kidney Injury, systemic mycosis			

Figure: 4

Mortality and severity of AKI

AKI staged by AKIN

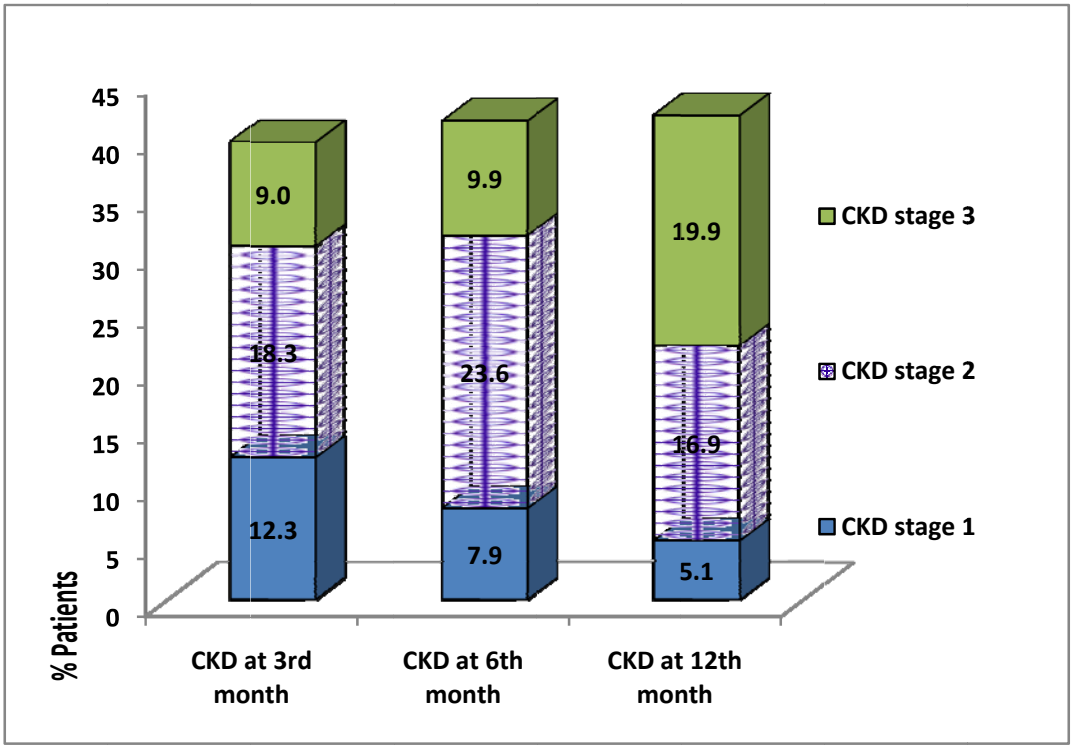


Survival time from HSCT (Days)

Progression to chronic kidney disease:

A total 57(41.9%) progressed to chronic kidney disease at the end of 12months follow up on adjustment with mortality (n=143); none of the patients had eGFR of <30ml/min and CKD was preceded by Acute kidney injury in all cases.

FIGURE 5: Progression to CKD



DISCUSSION

In our study we assessed post HSCT non-renal complications, incidence of AKI, possible causes of AKI, risk factors for AKI and outcome of patients that is, mortality. In addition, agreement between two commonly used criteria and staging of AKI (RIFLE, AKIN) was evaluated. Finally, independent predictors of AKI and mortality were analysed. A subset of patients with follow up data was analysed for development chronic kidney disease consequent to AKI.

Post HSCT complications (non renal):

We analysed post transplant complications as, infectitious and non-infectious complications. Among the infections, blood culture positive bacterial septicemia was observed in 40%. This was in contrast to the study by Saple et al [204] wherein only 15-20% bacterial sepsis was reported. The incidence of invasive fungal infections has been reported to be 10%-20% by Hovi L et al where as we observed 52% had suspected systemic mycosis. Nichols WG observed CMV incidence of 30% in seronegative patients however in our study, a relatively higher incidence of CMV (39.4%) was observed.

The above observations clearly indicate a significant higher rate of infections in our population compared to western studies.

Among the non infectious complications, various studies have demonstrated oral mucositis in about 75% of patients [207,208,209] compared to only 30% observed in our study as only only severe mucositis were documented.

GVHD was seen in 35% compared to 25-75% studies by Kiss and Lee et al [210,211].

Post HSCT complications (renal):

Incidence: Following HSCT, Acute Kidney Injury (AKI) is a very common problem which strongly limits patient's long-term survival. As shown in this study, 72% of the patients AKI, of which about half of them had stage I, II AKI.

As mentioned in previous studies, the incidence of renal dysfunction ranged from 56% to 92% in cohorts of patients receiving allogenic HSCT, with 20% requiring dialysis [212-216].

Our study showed that the incidence of renal dysfunction was similar to that in previous studies but only about 2% received dialysis support.

Risk Factors: Majority of the patients are between 22.0 ± 14.5 age group. This differs from study by Parikh et al where majority of the patients were 38.7 ± 9.7 .This low mean age in our study indicates that our study group was at lower risk of developing AKI.

Conditioning regimen, hepatic SOS, high risk malignancy, sepsis and GVHD were significant risk factors of AKI in a study by Zheng Ping et al [217], in comparison we found older age, matched unrelated donor (MUD), total body irradiation, Amphotericin B use and cyclosporine Level at 1st week were significant risk factors for AKI ,but not any infectious or immune related complications.

Besides infections and immune mediated complications, agents used to treat bacteraemia and sepsis (e.g. Gentamicin, Colistin, and Amphotericin B) may also be related to nephrotoxicity in our setting. Although much emphasis has been laid on reducing the exposure to or dose of cyclosporine to prevent renal injury, various studies have speculated that prevention of aGVHD and cGVHD or altering the cytokine and inflammatory response of GVHD will do more to reduce the incidence of renal injury following HSCT. The incidence of GVHD in our study is comparable with that of previous studies mentioned [218] .Our studies have demonstrated that the usual optimal cyclosporine levels (C0) for non haematological indications did not contribute to renal dysfunction and only a C0 of more than 301±158 ng/ml was significant risk factor for AKI.

AKI is a very common complication of HSCT patients on the ICU and has been reported to be associated with sepsis. In our study too, admission to the ICU was associated with AKI in this study. All AKIN stage 3 patients requiring dialysis had ICU admission.

Onset of AKI: Median duration of onset of AKI was 17 days with range of 2,290 days post HSCT, compared to 40 days and 33 days in a study by Kersting and Zheng Ping [217,218] et al respectively. The association of earlier onset of AKI in patients of SOS as reported by Kersting et al was not observed in our study but more severe degree of AKI (AKIN STAGE 3, RIFLE failure) occurred earlier in the post transplant period i.e 14 days (2,100). This earlier onset of AKI may be related to increased incidence of sepsis which mostly occurred in early post HSCT period.

Independent risk factors of AKI: In contrast to the study as by Kersting et al which reported that hypertension before transplantation as the only predictor for AKI, we found older age, and TBI as independent risk factors for AKI.

Table 27: Independent risk factors of AKI

Risk factors	Kersting et al	Parikh et al	Zheng Ping et al	Our study
	Hypertension	SOS	GVHD	Older age, TBI

TBI-Total Body Irradiation, GVHD-Graft versus host disease, SOS-Sinusoidal occlusive disease, AKI- Acute Kidney Injury

AKIN versus RIFLE criteria: Total of 149 (72.9%) patients had 170 episodes of AKI by AKIN criteria, average number of episodes per patient were 1.27. As expected AKIN classification was more sensitive in diagnosing AKI, the incidence of AKI by RIFLE and AKIN criteria was 69% and 72.9% respectively. There was a good correlation between the two criteria. About half of these AKI have moderate to severe degree of renal dysfunction (stage 2,3 AKIN) similar to study by Kersting et al wherein, severe renal failure was defined as doubling of the serum creatinine but not by current AKI classification.

MORTALITY:

Overall 34% mortality was observed at the end of study period, median time of death was 78 days (range; 0,801). Consistent with the increased risk of infections in HSCT patient's commonest cause of death was septicemia, followed by GVHD and relapse of primary disease.

AKI and mortality: Various studies have reported that renal dysfunction after HSCT contributed to patient mortality .In present study, mortality at the end of 6 months was 25.5% compared to 58% observed by Parikh et al [213].This difference could be attributed to lower mean age and co morbidities in our study group.

AKI was a significant risk factor for mortality. Other factors which were significant were older age, MUD, blood culture positive sepsis, CMV viremia, BKV viruria and SOS.

SOS, AKI, Sepsis, Systemic mycosis were independent predictors of mortality on multivariate analysis. To conclude, renal dysfunction, coupled with sepsis is much associated with high

risk of mortality in recipients after either allogenic HSCT, which has ominous implications for survival rate of the patients.

PROGRESSION TO CHRONIC KIDNEY DISEASE:

There was a paucity of data available in stem cell transplantation regarding progression to CKD. We observed that about one third progressed to CKD and majority of them had stage 3 CKD and none of them developed stage 4, stage 5 CKD.

Table 28: Summary of studies on AKI in allogenic myeloablative HSCT

Study	**Zager**	**Gruss**	**Ratanath arathorn**	**Letourne au**	**Nash**	**Parikh**	**Our study**
Study period	1986	1982–91	1993–94	1994–98	1995–96	1995–00	2010–11
Country	USA	Spain	USA	Canada	USA	USA	India
Total Patients	272	275	332	57	180	88	203
AKI in HSCT	54%	36%	76%	79%	80%	69%	72.9%
Death in patients without AKI	17%	18%	20%	60%	30%	40%	20%
Death in patients with AKI	58%	46%	43%	88%	42%	66%	40%
Death in patients with dialysis	84%	89%	95%	100%	94%	83%	80%

CONCLUSIONS

1. Incidence of AKI in HSCT patients: 72% by AKIN

2. About 50% of AKI was in stage 1

3. There is an excellent agreement between AKIN & RIFLE criteria.

4. Most of the AKI occurs in 2-3 weeks post HSCT

5. The independent risk factors of AKI: Older age and TBI.

6. SOS, Amphotericin, Sepsis, Systemic mycosis - independent risk factor for mortality

7. More than one third of patients progressed to develop CKD, of which majority were in CKD stage 3

8. AKIN valid in HSCT patients.

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PROFORMA

Case Number: **Month:** **Entered By:**

1. Serial no
2. Name
3. Age
4. Sex: Female -0 / Male -1
5. State / Country:
6. Height
7. Weight
8. BSA
9. DM No – 0 / Yes – 1
10. Hypertension No – 0 / Yes –1
11. CAD No – 0 / Yes – 1
12. Native disease:
13. Allogenic -0 Autologous HSCT-1

Myeloablative	Nonmyeloablative

13. Donor Name:
14. Donor Hosp Num:
15. Donor Age:
16. Donor Sex: Female -0 / Male -1
17. Donor Relation: Children - 4 / Parent -3 / Sibling - 2 / Spouse -1 / Others – 0(If Others – mention what)

- HLA Match:
- A: 0 / 1 / 2
- B: 0 / 1 / 2
- DR: 0 / 1 / 2
- DQ: 0 / 1 / 2

18. Nephrotoxic drugs received: No – 0 / Yes –1

Amphotericin	Vancomycin	Gentamycin	Other

19. Conditioning regimen: TBI+ Cyclophosphamide-1,

TBI+ Etopside-2,

Busulfan and cyclophosphamide-3,

Busulfan with fludarabine

20. TBI Dose

21. Prophylaxis against GVHD -CO -1/ Tac-2/ MTX-3

22. Infection prophylaxis: No – 0 / Yes –1

Fluconazole No – 0 / Yes –1,

Acyclovir No – 0 / Yes –1,

Trimethoprim –sulphamethoxazole No – 0 / Yes –1

23. Steroid dose

Steroid dose	Date from	Date to	Days	Equvivalent of Prednisolone (mg)	Cumulative Prednisolone dose (mg)

24. Cyclosporin: No -0 / Yes -1

25. CsA dose:

Cyclosporin Dose/day (mg)	Date from	Date to	Days	C0	C2	Date of C0/C2	Cumulative dose (mg)

m

26. Tacrolimus: No -0 / Yes -1

27. Starting dose of tacrolimus

28. Tac started on how many days before transplant?

29. Tac Level and Dose:

Tacrolimus Dose/day (mg)	Date from	Date To	Tacrolomis trough (ng/ml)	Date of Tac trough	Cumulative Tac dose (mg)

30.Total cumulative Tacrolimus dose at 1yr:

Date	Day	Wgt	MAP	CVP	INTK	O.PT	CR	GFR	UR	Na	K	HCO3	Ca	P	UA	

31. Hemogram

Date	Day	HB	TC	DC	P/S	PLT	LDH

32. Total No of Infective Episodes in the patient in the Post Tx period

33. No of episodes of Respiratory Tract Infection with Date

Date	Type of infection URI – 1 / Sinusitis – 2 / LRI – 3 / Lung Abscess – 4 / Empyema – 5 / Others – 6	Pathogen identified Neg – 0 / Pos – Bacteria – 1 / Fungus – 2 / PCP-3 / CMV - 4	Organism in blood c/s Neg – 0 / Pos - 1	Treatment antibiotic	Duration of treatment

n

34.UTI

Episodes➔	**1**	**2**	**3**	**4**	**5**	**6**
Urine C/S – number of colonies						
Urine C/S –organism **Bacteria-1** **Fungus - 2**						
Urine C/S –specify organism						
Rise in s.creat . No / if yes then to what level?						
Blood C/S - Organism						
Rx - antibiotic						
Duration (days)						
Classification: **Asymptomatic Bacteruria – 1**(No symptoms and not treated) **Cystitis/ Lower UTI – 2** (Symptoms local with or without fever and treated) **Acute Pyelonephritis – 3** (Febrile UTI with elevation of serum creatinine by a min of 0.2mg/dl without Blood c/s being positive) **Urosepsis – 4** (Blood c/s having same organism as urine c/s)						

o

35. Sepsis: No -0 / Yes -1

Date	Focus of infection UTI-1 / lri-2 / Skin & soft tissue – 3 / Endocaridum – 4 / Bone – 5 / Others (mention)	Organism in blood c/s	Treatment antibiotic	Duration of treatment

36.Other Bacterial Infections: No/Yes-1,2,3,4….episodes

Date	Focus of infection UTI-1 / lri-2 / Skin & soft tissue – 3 / Endocaridum – 4 / Bone – 5 / Others (mention)	Source of culture	Organism in c/s	Treatment antibiotic	Duration of treatment

37.VZV: No -0 / Herpes zoster -1 / Chicken Pox -2

38.Date:

39.Treatment Given:

40.HSV: No -0 / Yes -1 (if yes PCR)

41.LFT and PT ,APTT:

Date	Day	S.B	TP	SA	SGOT	SGPT	PT	APTT

43.Urine analysis:

Date	Day	Nitrate	Protein	RBC	WBC	UP/UC	Eosinophil	HBnuria

44.Hemodialysis:

Date	Day	Uremia	Acidosis	Hyperkalemia	V.overload	Hyperuricemia

Date	Day	SLED/regular	Heparin	access	Complications	Duration

45.ICU care:

Day	From	to	Number of days	Indication

46.VOD:

Date	Day	VOD(I-III)	Intervention

47.Mucositis

Date	Day	Mucosits (i-iv)	TPN

48. GVHD:

Date	Day	Skin (i-iii)	Liver (I-iii)	GIT(i-iii)	Intervention

q

UID	AKI yes /nc	state	Zone	Hosp ID	sex	age	death	Survival dt	Dt of LFU	dt death	cause of d	ht	wgt	bsa	b.grou	RH	dm	pre-ht	post-	CAD	u.disease
859	AKI	Rajasthan	4	575869C	Female	16	no	16.04.12	16.04.2012	.		145	33	1.17	B	POSTIVE	Not know	0	0	0	Thalassemia
860	AKI	Rajasthan	4	452069D	Male	9	no	19.03.12	19.03.2012	.		111	17	0.73	O	POSTIVE	Not know	0	Yes	0	Thalassemia
861	AKI	GJ	4	249086D	Male	11	no	02.06.12	02.06.2012	.		128	23	0.92	A	POSTIVE	Not know	0	0	0	Thalassemia
862	NO AKI	AP	3	607088D	Male	20	no	06.12.12	06.12.2012	.		174	50	1.59	A	POSTIVE	Not know	0	0	0	Aplastic ane
863	NO AKI	kerala	3	465938D	Male	2	no	05.07.12	05.07.2012	.		64	6.5	0.32	B	POSTIVE	Not know	0	0	0	Wiskott ALD
864	AKI	UP	1	483616D	Female	52	no	29.10.12	29.10.2012	.		153	64	1.61	A	POSTIVE	Not know	0	0	0	CML
865	AKI	AP	3	207392C	Male	10	yes	06.03.10	.	06.03.10	sepsis,v	125	23	0.89	O	POSTIVE	Not know	0	0	0	Thalassemia
866	NO AKI	Oman	5	616661D	Female	7	no	08.03.10	08.03.2010	.		81	11	0.49	A	POSTIVE	Not know	0	0	0	ALL
867	AKI	kerala	3	602097D	Male	31	yes	04.08.10	.	04.08.10	sysmyco	163	68	1.73	A	POSTIVE	Not know	0	0	0	AML
868	AKI	WB	2	575339D	Male	46	no	24.05.12	24.05.2012	.		172	71	1.84	B	POSTIVE	Not know	0	Yes	0	Aplastic ane
869	AKI	KA	3	463024D	Male	47	no	21.06.12	21.06.2012	.		179	89	2.08	O	POSTIVE	Not know	0	Yes	0	CML
870	AKI	AP	3	505773D	Female	52	no	05.11.12	05.11.2012	.		154	65	1.63	O	POSTIVE	Not know	0	0	0	ALL
871	AKI	MH	4	291985D	Female	8	no	07.05.12	07.05.2012	.		116	18	0.77	O	POSTIVE	Not know	0	0	0	Thalassemia
872	AKI	Srilanka	5	619327D	Female	45	no	04.11.10	04.11.2010	.		157	57	1.57	B	POSTIVE	Not know	0	0	0	AML
873	AKI	AP	3	606551D	Male	30	no	29.11.12	29.11.2012	.		171	66	1.77	B	POSTIVE	Not know	0	0	0	Aplastic ane
874	AKI	kerala	3	487826D	Female	26	no	20.12.12	20.12.2012	.		149	63	1.57	O	POSTIVE	Not know	0	0	0	ALL
875	AKI	kerala	3	334171C	Male	15	no	13.12.12	13.12.2012	.		152	51	1.46	O	POSTIVE	Not know	0	0	0	Aplastic ane
877	AKI	UP	1	607417D	Male	29	no	04.04.11	04.04.2011	.		171	55	1.64	B	POSTIVE	Not know	0	0	0	AML
878	NO AKI	MH	4	271409D	Male	8	no	27.09.12	27.09.2012	.		113	13	0.67	A	POSTIVE	Not know	0	0	0	Thalassemia
879	AKI	AP	3	619264C	Female	23	no	10.12.12	10.12.2012	.		161	58	1.61	O	POSTIVE	Not know	0	Yes	0	Aplastic ane
880	AKI	WB	2	530345D	Male	48	yes	17.06.10	.	17.06.10	sysmyc	164	63	1.68	B	POSTIVE	Not know	0	0	0	CMML
881	AKI	Delhi	1	612836D	Male	21	no	16.02.12	16.02.2012	.		177	91	1.98	B	POSTIVE	Not know	0	0	0	ALL
882	AKI	AP	3	633221D	Male	39	no	05.11.12	05.11.2012	.		155	54	1.51	O	NEGAGI	Not know	0	0	0	Aplastic ane
883	NO AKI	KA	3	431214d	Male	4	no	05.07.12	05.07.2012	.		86	12	0.52	AB	POSTIVE	Not know	0	0	0	Thalassemia
884	AKI	CH	1	465204C	Female	10	no	24.12.12	24.12.2012	.		126	23	0.91	O	POSTIVE	Not know	0	0	0	Thalassemia
885	AKI	Rajasthan	4	413636D	Male	22	no	21.06.12	21.06.2012	.		177	66	1.84	B	POSTIVE	Not know	0	0	0	MDS
886	NO AKI	manipur	2	182812D	Male	7	no	09.07.12	09.07.2012	.		102	16	0.67	O	POSTIVE	Not know	0	0	0	Thalassemia
887	AKI	UP	1	660012D	Male	13	no	29.11.12	29.11.2012	.		147	37	1.24	B	POSTIVE	Not know	0	0	0	Aplastic ane
888	AKI	Bihar	1	541802D	Female	19	yes	03.03.12	.	03.03.12	sepsi	157	51	1.45	B	POSTIVE	Not know	0	0	0	ALL
889	AKI	kerala	3	656191D	Male	46	yes	03.05.10	.	03.05.10	sepsi	174	88	2.03	O	POSTIVE	Not know	0	0	0	Aplastic ane
890	NO AKI	WB	2	570966D	Female	9	no	01.11.12	01.11.2012	.		112	20	0.79	A	POSTIVE	Not know	0	0	0	Biphenotypi
891	AKI	TN	3	581739B	Male	15	no	16.08.12	16.08.2012	.		139	27	1.04	O	POSTIVE	Not know	0	0	0	Thalassemia
892	AKI	AP	3	663573D	Female	26	no	03.12.12	03.12.2012	.		150	47	1.4	B	POSTIVE	Not know	0	0	0	Aplastic ane
893	AKI	AP	3	422909C	Male	22	no	22.10.12	22.10.2012	.		160	66	1.7	O	POSTIVE	Not know	0	0	0	AML

894	AKI	kerala	3	644382D	Male	36	yes	10.09.10	.	10.09.10	GVHD	170	60	1.69	B	POSTIVE	Not known	0	0	0	AML
895	NO AKI	AP	3	551715D	Male	46	no	17.09.12	17.09.2012	.		162	56	1.61	B	NEGAGI	Not known	0	0	0	CML
896	NO AKI	MH	4	970022C	Male	11	no	26.04.12	26.04.2012	.		135	22	0.9	B	POSTIVE	Not known	0	0	0	Thalassemia
897	AKI	AP	3	125569D	Male	26	no	17.12.12	17.12.2012	.		165	62	0.8	B	POSTIVE	Not known	0	0	0	Aplastic ane
898	AKI	kerala	3	122477D	Male	6	no	05.07.12	05.07.2012	.		95	13	0.5	B	POSTIVE	Not known	0	0	0	Wiskott ALD
899	AKI	KL	3	728811C	Male	13	no	18.10.12	18.10.2012	.		144	48	1.36	B	POSTIVE	Not known	0	0	0	ALL
900	AKI	Jharkhand	1	296183D	Male	13	yes	10.09.10	.	17.07.10	mi	131	26	0.98	O	POSTIVE	Not known	0	0	0	Thalassemia
901	AKI	megalaya	2	450340D	Female	27	yes	01.03.11	.	01.03.11	sepsis	142	40	1.25	O	POSTIVE	Not known	Yes	Yes	Yes	CML
902	AKI	GJ	4	628280D	Male	27	yes	21.08.12	.	21.08.12	sysmy	178	83	2.02	A	POSTIVE	Not known	0	0	0	ALL
903	AKI	CH	1	678900D	Male	45	yes	13.09.10	.	13.09.10	GVHD	175	84	1.99	B	POSTIVE	Not known	0	Yes	0	Aplastic ane
904	AKI	kerala	3	649431D	Female	22	no	05.11.12	05.11.2012	.		166	58	1.64	O	POSTIVE	Not known	0	0	0	AML
905	AKI	CH	1	674227D	Female	38	yes	03.12.10	.	03.12.10	seps	160	47	1.45	B	POSTIVE	Not known	0	0	0	AML
906	AKI	kerala	3	469353D	Female	8	no	09.08.12	09.08.2012	.		126	26	0.96	A	POSTIVE	Not known	0	0	0	Aplastic ane
907	NO AKI	MH	4	329021D	Male	9	no	18.10.10	18.10.2010	.		119	23	0.86	O	POSTIVE	Not known	0	0	0	Thalassemia
908	AKI	PJ	1	667763D	Male	28	yes	19.10.11	.	19.10.11	relapse	117	65	1.81	B	POSTIVE	Not known	0	0	0	ALL
909	NO AKI	TN	3	576195D	Female	14	no	06.12.12	06.12.2012	.		141	34	1.16	B	POSTIVE	Not known	0	0	0	Aplastic ane
912	AKI	AP	3	620964D	Female	44	yes	01.12.10	.	01.12.10	seps	157	57	1.57	O	POSTIVE	Not known	0	0	0	ALL
913	AKI	Jharkhand	1	628977D	Male	5	yes	20.09.12	.	20.09.12	seps	100	13	0.61	B	POSTIVE	Not known	0	0	0	MDS
914	AKI	kerala	3	660484D	Male	19	yes	04.10.10	.	04.10.10	GVHD	175	46	1.55	O	POSTIVE	Not known	0	0	0	Aplastic ane
915	NO AKI	Delhi	1	515227D	Male	8	no	22.07.12	22.07.2012	.		114	20	0.79	B	POSTIVE	Not known	0	0	0	Thalassemia
916	AKI	UP	1	677941D	Female	17	yes	15.11.10	.	15.11.10	seps	157	38	1.31	B	POSTIVE	Not known	0	0	0	ALL
917	AKI	kerala	3	711879D	Male	41	yes	02.10.11	.	02.10.11	seps	171	54	1.63	AB	POSTIVE	Yes	0	0	0	CML
918	NO AKI	MH	4	692401D	Female	3	yes	14.09.11	.	14.09.11	GVHD	75	8.1	0.4	A	POSTIVE	Not known	0	0	0	Thalassemia
919	AKI	AP	3	655885D	Male	33	yes	10.08.10	.	10.08.10	seps	165	65	1.72	B	POSTIVE	Not known	0	0	0	Aplastic ane
920	AKI	Srilanka	5	725651D	Female	36	no	17.12.12	17.12.2012	.		150	47	1.39	O	POSTIVE	Not known	0	0	0	CML
921	AKI	AP	3	505873D	Male	2	yes	09.09.10	.	09.09.10	VOD	75	7.2	0.3	O	POSTIVE	Not known	0	0	0	Sevre combi
922	AKI	HR	1	678537D	Female	7	yes	16.10.10	.	16.10.10	GVHD	117	22	0.84	B	POSTIVE	Not known	0	0	0	ALL
923	NO AKI	CH	1	481854D	Female	8	no	16.09.12	16.09.2012	.		117	20	0.8	A	POSTIVE	Not known	0	0	0	Thalassemia
924	AKI	CH	1	292917D	Female	41	no	16.12.12	16.12.2012	.		155	56	1.54	A	POSTIVE	Not known	0	0	0	AML
925	NO AKI	CH	1	481855D	Female	10	no	11.08.12	11.08.2012	.		124	22	0.88	O	POSTIVE	Not known	0	0	0	Thalassemia
926	AKI	AP	3	707078D	Male	39	yes	09.02.12	.	09.02.12	GVHD	166	61	1.68	A	POSTIVE	Not known	0	0	0	AML
927	AKI	MP	1	534002D	Male	13	no	11.10.12	11.10.2012	.		130	26	0.98	O	POSTIVE	Not known	0	0	0	Thalassemia
928	AKI	AP	3	678207D	Female	26	yes	20.09.10	.	20.09.10	sysmy	153	50	1.45	O	POSTIVE	Not known	0	0	0	Aplastic ane
929	AKI	TN	3	067558D	Male	31	no	18.10.12	18.10.2012	.		186	88	2.13	O	POSTIVE	Not known	0	0	0	Aplastic ane
930	NO AKI	UP	1	682483D	Female	30	no	20.08.12	20.08.2012	.		152	39	1.3	A	POSTIVE	Not known	0	0	0	Aplastic ane

931	AKI	MP	1	252693C	Male	12	yes	10.10.10	.	10.10.10	sysmy	139	31	1.11	O	POSTIVE	Not known	0	0	0 Thalassemia
933	AKI	TN	3	544133D	Male	32	no	12.11.12	12.11.2012	.		172	44	1.49	O	POSTIVE	Not known	0	0	0 Aplastic ane
934	AKI	Orissa	1	646571D	Male	38	no	03.12.12	03.12.2012	.		160	61	1.63	O	POSTIVE	Not known	0	Yes	0 Biphenotypi
935	NO AKI	kerala	3	755366D	Female	14	no	10.09.12	10.09.2012	.		146	38	1.25	A	POSTIVE	Not known	0	0	0 AML
936	AKI	AP	3	704717D	Female	27	no	07.06.12	07.06.2012	.		163	60	1.64	O	POSTIVE	Not known	0	0	0 AML
937	AKI	Srilanka	5	704564D	Male	22	yes	24.11.10	.	24.11.10	GVHD	176	51	1.62	A	POSTIVE	Not known	0	0	0 AML
938	AKI	Maldives	5	573907c	Male	16	no	22.06.12	22.06.2012	.		145	31	1.14	O	POSTIVE	Not known	0	0	0 Thalassemia
939	AKI	AP	3	775851D	Female	38	no	15.10.12	15.10.2012	.		149	60	1.54	O	POSTIVE	Not known	0	0	0 Aplastic ane
940	AKI	kerala	3	744792D	Female	34	no	05.11.12	05.11.2012	.		158	45	1.42	B	POSTIVE	Not known	0	0	0 AML
941	AKI	AP	3	593040D	Male	48	yes	20.01.11	.	20.01.11	GVHD	170	67	1.77	O	POSTIVE	Not known	0	0	0 CMML
942	NO AKI	AP	3	657682D	Male	28	yes	08.03.11	.	08.03.11	seps	163	48	1.51	O	POSTIVE	Not known	0	0	0 CML
943	NO AKI	AP	3	703699D	Male	48	no	13.09.12	13.09.2012	.		170	73	1.84	O	POSTIVE	Not known	0	0	0 CML
944	AKI	kerala	3	718141D	Male	46	no	05.11.12	05.11.2012	.		159	62	1.63	A	POSTIVE	Not known	0	0	0 AML
945	AKI	kerala	3	489024D	Male	7	no	03.12.12	03.12.2012	.		119	19	0.8	O	POSTIVE	Not known	Yes	0	0 fANCONI
946	NO AKI	TN	3	430745D	Female	6	no	27.12.12	27.12.2012	.		108	22	0.79	B	POSTIVE	Not known	0	0	0 MDS
947	AKI	PAK	5	445080c	Female	12	no	03.06.12	03.06.2012	.		130	27	0.99	O	POSTIVE	Not known	0	0	0 Thalassemia
948	NO AKI	GJ	4	909090B	Female	13	no	11.06.12	11.06.2012	.		143	30	1.1	B	POSTIVE	Not known	0	0	0 Thalassemia
949	AKI	TN	3	781371D	Male	30	no	17.12.12	17.12.2012	.		179	98	2.03	B	POSTIVE	Not known	0	0	0 AML
950	NO AKI	Bihar	1	671121D	Male	14	no	20.12.12	20.12.2012	.		157	57	1.57	A	POSTIVE	Not known	0	0	0 AML
951	AKI	kerala	3	760217D	Male	50	no	10.12.12	10.12.2012	.		172	90	1.9	O	POSTIVE	Not known	0	0	0 AML
952	AKI	TN	3	662815D	Male	9	yes	11.01.11	.	11.01.11	VOD	125	24	0.92	O	POSTIVE	Not known	0	0	0 Aplastic ane
953	AKI	TN	3	712230D	Female	43	no	13.08.12	13.08.2012	.		157	68	1.69	B	POSTIVE	Not known	0	0	0 Aplastic ane
954	AKI	AP	3	771714D	Male	58	yes	25.01.11	.	25.01.11	unknown	156	64	1.64	O	POSTIVE	Not known	0	0	0 AML
956	AKI	TN	3	828259D	Male	50	yes	24.12.10	.	24.12.10	seps	182	30	2.02	O	POSTIVE	Not known	0	0	0 Aplastic ane
957	NO AKI	HR	1	778463D	Male	6	no	04.10.12	04.10.2012	.		107	16	0.69	A	POSTIVE	Not known	0	0	0 Thalassemia
958	AKI	UP	1	737263D	Male	15	no	29.10.12	29.10.2012	.		152	32	1.2	B	POSTIVE	Not known	0	0	0 Aplastic ane
959	AKI	Rajasthan	4	828456D	Male	44	yes	21.09.11	.	21.09.11	sysmy	170	70	1.81	O	POSTIVE	Not known	0	0	0 CML
960	AKI	ap	3	483568D	Female	14	no	25.06.12	25.06.2012	.		127	19	0.85	O	POSTIVE	Not known	0	0	0 ALL
962	NO AKI	kl	3	399415D	Male	5	no	29.11.12	29.11.2012	.		97	14	0.61	O	NEGAGI	Not known	0	0	0 Thalassemia
963	AKI	mp	1	300035D	Female	6	yes	28.01.11	.	28.01.11	seps	110	19	0.96	B	POSTIVE	Not known	0	0	0 Thalassemia
964	AKI	Ka	3	778164D	Male	16	no	19.01.12	19.01.2012	.		171	69	1.8	O	POSTIVE	Not known	0	Yes	0 ALL
965	AKI	mh	4	551185D	Male	3	yes	25.01.11	.	25.01.11	seps	79	10	0.46	B	POSTIVE	Not known	0	0	0 Wiskott ALD
966	AKI	KL	3	002514D	Male	46	no	13.12.12	13.12.2012	.		176	61	1.75	A	POSTIVE	Not known	0	0	0 NHL
967	AKI	maldives	5	385013D	Male	12	no	07.11.12	07.11.2012	.		138	41	1.24	A	POSTIVE	Not known	0	0	0 Thalassemia
968	NO AKI	gj	4	385329D	Female	24	yes	19.04.11	.	19.04.11	CMV	156	85	1.63	B	POSTIVE	Not known	0	0	0 MDS

969	AKI	mh	4	713123D	Male	10	no	12.11.12	12.11.2012	.		130	20	0.85	AB	POSTIVE	Not known	0	0	0 Thalassemia
970	AKI	kl	3	773911D	Male	36	no	17.12.12	17.12.2012	.		168	80	1.9	B	POSTIVE	Not known	0	0	0 ALL
971	NO AKI	kl	3	635354D	Female	23	no	22.10.12	22.10.2012	.		162	56	1.58	A	POSTIVE	Not known	0	0	0 Aplastic ane
972	NO AKI	bd	5	366323D	Male	4	no	30.07.12	30.07.2012	.		93	12	0.55	B	POSTIVE	Not known	0	0	0 Thalassemia
973	AKI	rj	4	870174D	Male	55	yes	17.06.11	.	17.06.11	GVHD	155	75	1.57	O	POSTIVE	Not known	0	0	0 AML
974	AKI	kl	3	332714D	Male	15	no	18.10.12	18.10.2012	.		150	49	1.42	B	POSTIVE	Not known	0	0	0 fANCONI
975	AKI	gj	4	772247D	Male	8	yes	13.03.11	.	13.03.11	DCMY	135	35	1.14	B	POSTIVE	Not known	0	0	0 ALL
976	NO AKI	tn	3	852416D	Male	17	yes	11.04.11	.	11.04.11	sysmy	174	63	1.75	O	POSTIVE	Not known	0	0	0 Aplastic ane
977	NO AKI	mh	4	859324D	Male	5	no	01.11.12	01.11.2012	.		104	15	0.65	A	POSTIVE	Not known	0	0	0 Aplastic ane
978	AKI	Rj	4	754178D	Female	28	yes	20.04.12	.	20.04.12	GVHD	161	57	1.6	O	POSTIVE	Not known	0	0	0 AML
979	NO AKI	delhi	1	715993D	Female	5	no	13.12.12	13.12.2012	.		91	15	0.6	B	POSTIVE	Not known	0	0	0 Thalassemia
980	AKI	pj	1	709066B	Female	21	no	17.12.12	17.12.2012	.		154	50	1.47	O	POSTIVE	Not known	0	0	0 Thalassemia
981	AKI	bd	5	772733D	Female	38	no	10.12.12	10.12.2012	.		152	65	1.62	A	POSTIVE	Not known	0	0	0 AML
982	AKI	kl	3	556416C	Female	24	no	29.11.12	29.11.2012	.		156	58	1.57	B	POSTIVE	Not known	0	0	0 ALL
983	NO AKI	wb	2	995353C	Female	4	yes	05.04.11	.	05.04.11	DAH	95	11	0.55	B	POSTIVE	Not known	0	0	0 Thalassemia
984	AKI	bd	5	782971D	Female	23	no	15.11.12	15.11.2012	.		59	.	1.6	A	POSTIVE	Not known	0	Yes	0 Aplastic ane
985	AKI	ka	3	838882D	Female	45	yes	23.12.11	.	23.12.11	relapse	158	59	1.6	A	POSTIVE	Not known	0	0	0 AML
987	AKI	up	1	762637D	Male	8	yes	04.10.11	.	04.10.11	gfaftfai	128	27	0.99	O	NEGAGI	Not known	0	0	0 Aplastic ane
988	NO AKI	mp	1	533372D	Female	11	no	18.10.12	18.10.2012	.		126	22	0.89	B	POSTIVE	Not known	0	Yes	0 Thalassemia
989	AKI	ap	3	727652D	Male	48	yes	09.06.11	.	09.06.11	GVHD	167	53	1.61	O	POSTIVE	Not known	0	0	0 CML
990	NO AKI	gj	4	421447d	Female	6	no	10.09.12	10.09.2012	.		118	16	0.75	AB	POSTIVE	Not known	0	0	0 Thalassemia
991	AKI	up	1	869183D	Male	36	yes	01.06.11	.	01.06.11	GVHD	164	57	1.6	B	POSTIVE	Not known	0	0	0 Aplastic ane
992	.	ap	3	444421C	Male	38	no	20.12.12	20.12.2012	.		169	57	1.65	A	POSTIVE	Not known	0	0	0 CML
994	AKI	wb	2	867664D	Male	16	no	30.04.12	30.04.2012	.		169	49	1.55	A	POSTIVE	Yes	0	0	0 Aplastic ane
995	NO AKI	uae	5	856566D	Male	6	no	13.12.12	13.12.2012	.		113	20	0.79	B	POSTIVE	Not known	0	0	0 ALL
996	AKI	ka	3	851912D	Female	20	yes	24.05.11	.	24.05.11	seps	155	49	1.45	O	POSTIVE	Not known	0	0	Yes Aplastic ane
997	AKI	mal	5	371555D	Female	14	no	28.06.12	28.06.2012	.		150	44	1.3	O	POSTIVE	Not known	0	0	0 Thalassemia
998	AKI	bihar	1	764872D	Male	38	yes	06.02.12	.	06.02.12	GVHD	175	76	1.91	AB	POSTIVE	Not known	0	0	0 CML
999	AKI	AP	3	403686d	Male	39	no	29.11.12	29.11.2012	.		162	69	1.74	B	NEGAGI	Not known	0	Yes	0 Aplastic ane
1000	NO AKI	delh	1	882729D	Female	30	yes	14.01.12	.	14.01.12	GVHD	148	60	1.73	A	POSTIVE	Not known	0	0	0 AML
1004	AKI	ap	3	802085D	Male	22	no	17.12.12	17.12.2012	.		165	72	1.79	O	POSTIVE	Not known	0	0	0 MDS
1005	AKI	dubai	5	929212D	Male	39	no	19.10.11	19.10.2011	.		158	62	1.63	A	POSTIVE	Not known	0	0	0 AML
1006	AKI	jh	1	857038D	Male	12	no	24.10.12	24.10.2012	.		127	23	.	O	POSTIVE	Not known	0	0	0 Aplastic ane
1007	AKI	GJ	4	420542D	Female	3	yes	24.05.11	.	24.05.11	CHF	134	27	1.02	B	POSTIVE	Not known	0	0	0 Thalassemia
1008	AKI	hr	1	867444D	Male	16	no	04.10.12	04.10.2012	.		148	34	1.2	B	POSTIVE	Not known	0	Yes	0 Thalassemia

1009	NO AKI	mp	1	186648B	Female	19	no	24.09.12	24.09.2012	.		146	40	1.28	O	POSTIVE	Not known	0	0	0 Thalassemia
1010	NO AKI	ap	3	547890D	Female	11	no	15.11.12	15.11.2012	.		137	37	1.18	A	POSTIVE	Not known	0	0	0 fANCONI
1011	NO AKI	tn	3	618451b	Male	17	yes	11.12.11	.	11.12.11	seps	138	34	1.1	B	POSTIVE	Not known	0	0	0 Thalassemia
1012	.	pakistan	5	553671D	Male	11	no	03.07.12	03.07.2012	.		125	22	0.89	AB	POSTIVE	Not known	0	0	0 Thalassemia
1013	.	up	1	811799D	Male	6	no	19.11.12	19.11.2012	.		99	16	0.64	O	POSTIVE	Not known	0	0	0 Thalassemia
1014	AKI	ap	3	263735D	Female	36	yes	29.08.11	.	29.08.11	GVHD	155	57	1.56	B	NEGAGI	Not known	0	0	0 AML
1015	AKI	g	4	801692D	Male	25	yes	29.09.11	.	29.09.11	sysmy	167	53	1.58	B	POSTIVE	Not known	0	0	0 ALL
1016	.	kl	3	850858D	Male	18	yes	02.11.12	.	02.11.12	sysmy	176	60	1.78	B	NEGAGI	Not known	0	0	0 AML
1017	AKI	podicherry	3	900139D	Male	49	yes	15.09.11	.	15.09.11	GVHD	163	55	1.59	AB	POSTIVE	Not known	0	0	0 Aplastic ane
1018	AKI	kl	3	978588C	Female	9	no	05.11.12	05.11.2012	.		134	28	1.03	A	POSTIVE	Not known	0	Yes	0 ALL
1019	NO AKI	wb	2	245101D	Female	4	no	13.12.12	13.12.2012	.		97	13	0.59	O	POSTIVE	Not known	0	0	0 Thalassemia
1020	AKI	ap	3	865247D	Female	1	no	20.12.12	20.12.2012	.		75	11	0.45	O	POSTIVE	Not known	0	0	0 ALL
1021	NO AKI	up	1	509928D	Female	10	no	17.09.12	17.09.2012	.		130	24	0.94	B	NEGAGI	Not known	0	0	0 Thalassemia
1022	NO AKI	gj	4	962119D	Female	4	no	15.11.12	15.11.2012	.		95	13	0.58	O	POSTIVE	Not known	Yes	0	0 Aplastic ane
1023	AKI	kl	3	900095D	Female	7	yes	10.04.12	.	10.04.12	GVHD	144	22	0.88	O	POSTIVE	Not known	0	0	0 ALL
1025	AKI	ap	3	817630D	Male	21	no	17.12.12	17.12.2012	.		172	77	1.9	A	POSTIVE	Not known	0	0	0 Aplastic ane
1026	NO AKI	ch	1	539365D	Female	5	no	20.12.12	20.12.2012	.		96	14	0.61	O	POSTIVE	Not known	0	0	0 Thalassemia
1027	NO AKI	mh	4	653219D	Female	8	no	22.10.12	22.10.2012	.		117	19	0.79	B	NEGAGI	Not known	0	0	0 Thalassemia
1028	AKI	bd	5	867863D	Male	30	yes	24.07.12	.	24.07.12	GVHD	165	49	1.53	O	POSTIVE	Not known	0	0	0 ALL
1029	AKI	ap	3	036911D	Male	11	no	29.11.12	.	29.11.12	sysmy	162	45	1.45	O	POSTIVE	Not known	0	0	0 fANCONI
1030	AKI	arp	2	835360D	Female	17	yes	26.11.11	.	26.11.11	sysmy	152	34	1.52	AB	POSTIVE	Not known	0	0	0 ALL
1031	NO AKI	gj	4	920594D	Male	3	no	10.03.12	10.03.2012	.		84	8.5	0.44	B	POSTIVE	Not known	0	Yes	0 CMML
1032	AKI	ap	3	924770D	Male	27	no	08.11.12	08.11.2012	.		177	75	1.92	A	POSTIVE	Not known	0	Yes	0 AML
1033	AKI	srilanka	5	987025D	Male	17	no	16.10.12	16.10.2012	.		168	69	1.78	O	POSTIVE	Not known	0	0	0 AML
1034	AKI	ka	3	863857D	Male	33	yes	08.10.11	.	08.10.11	SOS	169	70	1.82	A	POSTIVE	Not known	0	0	0 AML
1037	AKI	up	1	737293D	Female	34	yes	14.09.11	.	14.09.11	sysmy	147	67	1.59	B	POSTIVE	Not known	0	0	0 MDS
1038	AKI	ka	3	749214D	Male	3	no	17.12.12	17.12.2012	.		90	13	0.58	O	POSTIVE	Not known	0	0	0 Thalassemia
1039	AKI	ap	3	955109D	Female	15	no	12.11.12	12.11.2012	.		160	47	1.47	O	NEGAGI	Not known	0	0	0 Aplastic ane
1040	NO AKI	tn	3	900218D	Male	40	yes	30.09.11	.	30.09.11	seps	176	60	1.73	B	POSTIVE	Not known	0	0	0 AML
1041	AKI	tn	3	895137D	Female	36	yes	05.11.11	.	05.11.11	seps	165	63	1.69	B	POSTIVE	Not known	0	0	0 CML
1042	AKI	kl	3	931646D	Male	18	no	17.12.12	17.12.2012	.		175	62	1.75	O	POSTIVE	Not known	0	Yes	0 Aplastic ane
1043	AKI	up	1	388670D	Female	12	no	13.09.12	13.09.2012	.		144	31	1.14	B	POSTIVE	Not known	0	0	0 Thalassemia
1044	AKI	JH	1	891211D	Female	39	yes	22.11.11	.	22.11.11	sysmy	156	64	1.63	B	POSTIVE	Not known	0	0	0 AML
1045	NO AKI	mh	4	463223D	Female	8	no	26.11.12	26.11.2012	.		106	14	0.65	B	POSTIVE	Not known	0	0	0 Thalassemia
1046	AKI	tn	3	902811D	Male	39	no	20.12.12	20.12.2012	.		173	73	1.87	AB	POSTIVE	Not known	0	0	0 CML

1047	NO AKI	gj	4	796402D	Male	20	no	19.04.12	19.04.2012	.		147	35	1.22	O	POSTIVE	Not known	0	0	0 Thalassemia
1048	AKI	ap	3	993945D	Male	35	no	13.12.12	13.12.2012	.		162	61	1.65	B	POSTIVE	Not known	0	0	0 AML
1049	AKI	gj	4	650095D	Male	9	no	30.04.12	30.04.2012	.		123	20	0.84	B	POSTIVE	Not known	0	0	0 Thalassemia
1050	AKI	or	1	370562D	Male	28	no	17.12.12	17.12.2012	.		159	80	1.88	B	POSTIVE	Not known	0	0	0 ALL
1051	NO AKI	tn	3	000694F	Female	59	yes	01.11.11	.	01.11.11	seps	156	95	1.67	A	POSTIVE	Not known	0	0	0 MDS
1052	AKI	srilanka	5	498267D	Male	6	no	17.12.12	17.12.2012	.		102	16	0.64	B	POSTIVE	Not known	0	0	0 Thalassemia
1054	NO AKI	ch	1	878863D	Male	7	no	20.12.12	20.12.2012	.		114	20	0.8	O	POSTIVE	Not known	0	0	0 Thalassemia
1055	AKI	kl	3	246961D	Female	7	no	01.11.12	01.11.2012	.		116	21	0.8	B	POSTIVE	Not known	0	0	0 ALL
1056	AKI	ap	3	995419D	Male	29	yes	04.01.12	.	04.01.12	sysmy	165	58	1.64	B	POSTIVE	Not known	0	0	0 Aplastic ane
1058	AKI	oman	5	046904F	Female	22	no	24.04.12	24.04.2012	.		153	45	1.37	O	POSTIVE	Not known	0	0	0 Thalassemia
1059	AKI	srilan	5	980469D	Male	10	yes	27.08.12	.	27.08.12	STOKE	130	25	0.96	A	NEGAGI	Not known	0	0	0 AML
1060	AKI	ch	1	898790D	Male	9	no	03.12.12	03.12.2012	.		116	19	0.8	O	POSTIVE	Not known	0	0	0 Thalassemia
1061	AKI	kl	3	024060F	Female	24	no	13.12.12	13.12.2012	.		158	44	1.41	B	POSTIVE	Not known	0	0	0 AML
1062	AKI	hr	1	933685D	Female	7	no	04.10.12	04.10.2012	.		107	19	0.74	O	POSTIVE	Not known	0	0	0 Thalassemia
1063	AKI	oman	5	046905F	Male	21	no	23.10.12	23.10.2012	.		.	.	1.31	O	POSTIVE	Not known	0	0	0 Thalassemia
1065	AKI	bd	5	948434D	Male	28	no	08.11.12	08.11.2012	.		153	47	1.42	B	POSTIVE	Not known	0	0	0 AML
1066	NO AKI	kl	3	435204D	Male	32	no	17.12.12	17.12.2012	.		167	82	1.91	O	POSTIVE	Yes	0	0	0 CML
1067	AKI	hr	1	775462D	Male	9	yes	22.02.12	.	22.02.12	GVHD	125	25	0.93	B	POSTIVE	Not known	0	0	0 Thalassemia
1068	AKI	tn	3	946420D	Male	39	yes	28.02.12	.	28.02.12	GVHD	172	69	1.81	AB	POSTIVE	Not known	0	0	0 NHL
1069	AKI	kl	3	211328D	Female	30	no	19.11.12	19.11.2012	.		180	76	1.95	B	POSTIVE	Not known	0	0	0 Aplastic ane
1070	AKI	ka	3	058502F	Female	42	yes	15.04.12	.	15.04.12	GVHD	160	67	1.7	O	POSTIVE	Not known	0	0	0 AML
1071	AKI	ap	3	887622D	Male	25	no	01.11.12	01.11.2012	.		168	52	1.51	A	POSTIVE	Not known	0	0	0 Aplastic ane
1072	NO AKI	ch	1	655455D	Male	7	yes	29.01.12	.	29.01.12	GVHD	120	20	0.85	B	POSTIVE	Not known	0	0	0 Thalassemia
1073	AKI	up	1	957518D	Female	21	yes	16.01.12	.	16.01.12	GVHD	163	46	1.47	O	POSTIVE	Not known	0	0	0 PNH
1074	NO AKI	mh	4	643838D	Male	8	no	03.12.12	03.12.2012	.		119	20	0.82	B	POSTIVE	Not known	0	0	0 Thalassemia
1075	AKI	mp	1	936056B	Male	18	no	17.12.12	17.12.2012	.		149	52	1.45	O	POSTIVE	Not known	0	0	0 Thalassemia
1076	AKI	tn	3	007704F	Male	36	no	19.11.12	19.11.2012	.		170	70	1.85	AB	POSTIVE	Not known	0	0	0 ALL
1077	AKI	TN	3	357422D	Male	15	yes	24.06.10	.	24.06.10	seps	160	42	1.45	A	POSTIVE	Not known	0	Yes	0 Aplastic ane
1078	AKI	AP	3	475365D	Male	2	yes	17.04.10	.	17.04.10	seps	73	7.9	0.29	O	POSTIVE	Not known	0	0	0 Sevre combi

donor	D.bl.grou	D.Rh	relation	MUD	HLA match	HLAa	HLAb	HLAdr	HLAdq	mismatch	Match	Conditioning	TBi conditioni	MNC	
578203C	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	2.49	
463268D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	3.91	
602051D	AB	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	6.6	
615880D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	5.09	
465939D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Cyc	Non TBI cond	1.5	
602868D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	7.06	
615204D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	3.7	
622735D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulp+CYclo	Non TBI cond	12.8	
620803D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	5.1	
624355D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	5.45	
615846D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	3.58	
	O	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	TBI+cyclopho	TBi conditioni	2.94	
622485D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	3.22	
619329D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	4.6	
635455D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	3.79	
606836D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	8.68	
587404D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	5.38	
653217D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	3.08	
640215D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	8.29	
658809D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	4.8	
653286D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	4.16	
659618D	B	POSTIVE	Sibiling	related	5	1	0	0	0	1	mismatch	TBI+cyclopho	TBi conditioni	5.67	
659999d	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	5.8	
648901d	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	4.76	
394327D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Others	Non TBI cond	2.83	
	B	POSTIVE	MUD	matched unre	8	0	0	0	0	2	mismatch	Others	Non TBI cond	3.16	
477134D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	4.4	
670177D	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	8.56	
	O	POSTIVE	MUD	matched unre	8	0	0	0	0	2	mismatch	TBI+cyclopho	TBi conditioni	12.5	
678784D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	0	
682183D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	8.54	
809727B	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	4.08	
681788D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	5.9	
679612D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	4.9	

655994D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	8.7
677468D	B	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	10.29
351021D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	4.6
690550D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	5
673115D	B	POSTIVE	others	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	6.6
631033d	O	POSTIVE	Parent	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	7.95
296185D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	3.97
694167D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	4.56
	A	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	4.37
683253D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	5.69
697154D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	12.15
682159D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	8.08
511722D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Cyc	Non TBI cond	14.2
335404D	O	POSTIVE	Parent	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	4.4
715797D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	3.41
673286D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	7.33
635525D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	6.35
706133D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATG+BUSUL+	Non TBI cond	7.34
732003d	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	7.3
727622D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	8.59
	A	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	5.27
738822d	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	9.1
726138D	O	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	8.47
733172D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	0
726427D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	10.4
492385D	O	POSTIVE	Parent	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	8.88
	O	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	15.37
749189D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	5.6
746915D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	4.43
749186D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	3.4
709448D	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	5.25
668598D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	2.5
745846D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+FLUDARB	TBi conditioni	17.12
	O	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	ATG+BUSUL+	Non TBI cond	5.6
767614D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	8.07

770869D	B	POSTIVE	Parent	related	5	0	0	0	0	1	mismatch	Thio+Treo+Flı	Non TBI cond	1.62
901732C	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;ı	Non TBI cond	5.12
758568D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	8.1
758046D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	14.8
763450d	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	4.4
773064D	AB	POSTIVE	Sibiling	related	5	0	0	0	0	1	mismatch	Melphelan+Fl	Non TBI cond	14.1
786070D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Others	Non TBI cond	8.94
795636D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;ı	Non TBI cond	4.5
794214D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	16.8
	O	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	4.1
795887d	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	Non TBI cond	5.5
797096D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	2.07
	A	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	7.5
808976D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	12.7
816940D	A	NEGAGIVE	Parent	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	10.7
445091C	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Others	Non TBI cond	12.9
824504D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Others	Non TBI cond	4.91
790079D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Others	Non TBI cond	10.4
	O	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	5
774003D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	7.1
705128d	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	5.13
829261D	B	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	Cyclophos+f;ı	Non TBI cond	5.03
	O	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	10.7
831822d	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;ı	Non TBI cond	3.39
836157D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	4.63
839336D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	5.7
842072D	O	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	Non TBI cond	6.78
827505D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	14
846242D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	.
846179D	O	POSTIVE	Parent	related	8	0	0	0	0	2	mismatch	Thio+Treo+Flı	Non TBI cond	3.6
809564D	O	POSTIVE	Parent	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	2.04
551185D	A	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	Others	Non TBI cond	65.37
803983d	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	7.37
385028D	O	POSTIVE	Sibiling	related	8	0	0	0	0	2	mismatch	Thio+Treo+Flı	Non TBI cond	8.18
	O	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	Busulfan+Fluc	Non TBI cond	6.97

732906d	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+bu	Non TBI cond	5.6
794704D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	5.8
868027D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	5.88
366526D	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	6.76
870445D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	4.7
801997D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATG+CYCLOPI	Non TBI cond	7.7
	O	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	TBI+cyclopho	Non TBI cond	.
860930D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	7.87
878121D	B	POSTIVE	Parent	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	10.9
	O	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	Others	Non TBI cond	6.4
866780D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	5.5
872029D	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	7.92
877566D	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	3.81
	A	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	TBI+cyclopho	TBi conditioni	6.22
907794D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	3.7
890061D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	5.17
888244D	A	POSTIVE	Children	related	9	0	0	0	0	1	mismatch	Busulfan+Fluc	Non TBI cond	3.43
	B	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	ATAGAM+Bus	Non TBI cond	8.9
536314D	AB	POSTIVE	Parent	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	10.8
	A	POSTIVE	MUD	matched unre	9	0	0	0	0	0	mismatch	TBI+cyclopho	TBi conditioni	3.54
421884D	B	POSTIVE	Parent	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	8.06
894884D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	8.53
886583D	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	6.05
903891d	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	4.6
906443d	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	13.26
903538	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	8
917208D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	5
	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	3.75
921407D	B	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	4.8
900526d	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	3.8
924718D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	4.7
929634D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	3.9
926697D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	10.6
925244d	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	11.2
923049D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Flu	Non TBI cond	6.8

	O	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	TBI+cyclopho:	TBi conditioni	12.3
941370D	A	POSTIVE	Sibiling	related	9	0	0	0	0	1	mismatch	ATG+CYCLOPI	Non TBI cond	10.8
618494B	B	POSTIVE	Parent	related	9	0	0	0	0	1	mismatch	Thio+Treo+Fl	Non TBI cond	6.7
551139D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	10.5
811799D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	5.94
885852D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	4
	B	NEGAGIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	TBI+cyclopho:	TBi conditioni	17.28
	O	NEGAGIVE	MUD	matched unre	8	0	0	0	0	0	mismatch	Busulfan+Fluc	Non TBI cond	6.6
957440D	A	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	10.1
	O	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	TBI+cyclopho:	TBi conditioni	8.53
945624D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	7.21
	A	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	Busulfan+Fluc	Non TBI cond	5.4
509928D	AB	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	6.1
979475D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	9.85
	B	POSTIVE	MUD	matched unre	8	0	0	0	0	2	mismatch	TBI+cyclopho:	TBi conditioni	7.5
979676D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	TBi conditioni	2.69
966601D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	7.1
653219D	B	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	9.4
876916D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho:	TBi conditioni	4.9
980599D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	7.1
884175D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	10.3
	A	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	Busulfan+Cyc	Non TBI cond	9.45
989753D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	7.37
991898D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	4.83
	A	POSTIVE	MUD	matched unre	9	0	0	0	0	0	mismatch	Busulfan+Fluc	Non TBI cond	.
005440F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	5.9
775720D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	6.5
493131D	O	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	7.77
	O	NEGAGIVE	MUD	matched unre	9	0	0	0	0	0	mismatch	Busulfan+Fluc	Non TBI cond	4.2
017607F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho:	TBi conditioni	4.5
020555F	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	4.21
762456F	B	NEGAGIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	6.2
892399D	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	4.13
771743D	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	6.2
913213D	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho:	TBi conditioni	3.56

	B	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	Others	Non TBI cond	6.2
000174F	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	7.2
018707F	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	6.2
	B	POSTIVE	MUD	matched unre	9	0	0	0	0	1	mismatch	TBI+cyclopho	TBi conditioni	3.12
037946F	A	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	ATG+BUSUL+	Non TBI cond	2.7
037598F	B	POSTIVE	Parent	related	10	0	0	0	0	0	no mismatch	ATAGAM+Bus	Non TBI cond	4.2
879545F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	5.1
	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	3.2
048067F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	3.52
053218F	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	4.3
052522F	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	2.6
048526F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	4.57
036506F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	4.5
65179	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	7.3
053220F	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	13.6
069477F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Melphelan+Fl	Non TBI cond	4.94
065393F	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	3.8
827496D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	2.69
051605F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	3.42
072757F	B	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	3.14
083375F	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Busulfan+Fluc	Non TBI cond	7.08
081673F	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Cyclophos+f;u	Non TBI cond	8.7
083732F	B	POSTIVE	Parent	related	9	0	0	0	0	1	mismatch	Thio+Treo+Fl	Non TBI cond	3.49
087185F	B	POSTIVE	Parent	related	8	0	0	0	0	2	mismatch	ATAGAM+Bus	Non TBI cond	2.43
643839D	O	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	Thio+Treo+Fl	Non TBI cond	6.73
	O	POSTIVE	MUD	matched unre	10	0	0	0	0	0	no mismatch	ATG+CYCLOPI	Non TBI cond	3.9
099356F	AB	POSTIVE	Sibiling	related	10	0	0	0	0	0	no mismatch	TBI+cyclopho	TBi conditioni	0.34
	AB	POSTIVE	Parent	related	4	1	1	0	0	2	mismatch	TBI+FLUDARB	TBi conditioni	2.5
	O	POSTIVE	Parent	related	4	1	1	0	0	2	mismatch	ATAGAM+Bus	Non TBI cond	3.92

CD34	Dt of HSCt	ANC	PLT	GVH	infecti	penici	oralac	septrar	WB	pac	PRC	FFP	SIRS	SEPSIS	Sepsis cause	DT sepsis	ORG	Sysmycosis	provenmycos	amika
3.62	04.01.2010	18.01.2010	28.01.2010	3	Yes	Yes	Yes	Yes	0	0	0	0	Yes	Sirs without n	Undifferentia	04.01.2010		nosuspcion	culture negat	no
9.86	11.01.2010	29.01.2010	18.02.2010	3	Yes	Yes	Yes	Yes	0	7	77	0	Yes	Sirs without n	Undifferentia	14.01.2010		Fungalinfectic	culture negat	yes
2.45	18.01.2010	08.02.2010	14.02.2010	3	Yes	Yes	Yes	Yes	0	5	48	3	Yes	Sepsis	Bacterial	23.01.2010	nfgnb	nosuspcion	culture negat	yes
21.2	21.01.2010	.	.	3	Yes	Yes	Yes	Yes	0	3	12	0	Yes	Sirs without n	Undifferentia	27.01.2010		nosuspcion	culture negat	no
9.38	21.01.2010	.	.	3	Yes	Yes	Yes	Yes	0	6	48	0	Yes	Sepsis	Bacterial	14.02.2010	coag neg s	Fungalinfectic	culture negat	no
13.6	06.02.2010	11.02.2010	.	3	Yes	Yes	Yes	Yes	0	0	0	0	Yes	Sirs without n	Undifferentia	26.01.2010		Fungalinfectic	culture negat	no
8.89	01.02.2010	22.02.2010	.	3	Yes	Yes	Yes	Yes	0	5	40	0	Yes	Sirs without n	Undifferentia	14.02.2010		Fungalinfectic	culture negat	yes
43.2	03.02.2010	.	.	3	Yes	Yes	Yes	Yes	0	2	9	0	Yes	Sirs without n	Undifferentia	10.01.2010		Fungalinfectic	culture negat	yes
13.3	11.02.2010	25.02.2010	01.03.2010	3	Yes	Yes	Yes	Yes	0	2	4	0	Yes	Sirs without n	Undifferentia	16.02.2010		Fungalinfectic	culture negat	yes
4.95	26.02.2010	26.02.2010	.	3	Yes	Yes	Yes	Yes	0	3	8	0	Yes	Sirs without n	Undifferentia	11.02.2010		Fungalinfectic	culture negat	no
9.3	15.02.2010	04.03.2010	11.03.2010	3	Yes	Yes	Yes	Yes	0	4	14	0	Yes	Sirs without n	Undifferentia	20.02.2010		Fungalinfectic	culture negat	yes
4.55	19.02.2010	06.03.2010	23.03.2010	3	Yes	Yes	Yes	Yes	0	5	44	0	Yes	Sepsis	Bacterial	04.04.2010	coag neg s	Fungalinfectic	culture negat	yes
15.7	22.02.2010	.	.	3	Yes	Yes	Yes	Yes	0	24	4	0	Yes	Sirs without n	Undifferentia	26.02.2010		nosuspcion	culture negat	yes
34.1	25.02.2010	01.03.2010	15.03.2010	3	Yes	Yes	Yes	Yes	0	5	29	0	Yes	Sirs without n	Undifferentia	26.02.2010		Fungalinfectic	culture negat	no
5.76	26.02.2010	.	.	3	Yes	Yes	Yes	Yes	0	3	14	0	Yes	Sirs without n	Undifferentia	25.02.2010		nosuspcion	culture negat	yes
29.2	11.03.2010	26.03.2010	26.03.2010	3	Yes	Yes	Yes	Yes	0	2	2	0	Yes	Sirs without n	Undifferentia	15.03.2010		nosuspcion	culture negat	yes
7.54	12.03.2010	.	.	3	Yes	Yes	Yes	Yes	0	2	6	0	Yes	Sirs without n	Undifferentia	15.03.2010		Fungalinfectic	culture negat	yes
7.2	18.03.2010	02.04.2010	06.04.2010	3	Yes	Yes	Yes	Yes	0	2	6	3	Yes	Sirs without n	Undifferentia	27.03.2010		Fungalinfectic	culture negat	yes
24.4	22.03.2010	.	.	3	Yes	Yes	Yes	Yes	0	3	24	0	Yes	Sirs without n	Undifferentia	26.03.2010		nosuspcion	culture negat	yes
11.4	31.03.2010	16.04.2010	23.04.2010	3	Yes	Yes	Yes	Yes	0	6	46	0	Yes	Sepsis	Bacterial	13.04.2010	enterobact	Fungalinfectic	culture negat	no
9.6	31.03.2010	12.04.2010	18.04.2010	3	Yes	Yes	Yes	Yes	0	6	10	0	Yes	Sepsis	Bacterial	30.03.2010	coag neg s	Fungalinfectic	culture negat	yes
9.6	01.04.2010	17.04.2010	19.04.2010	3	Yes	Yes	Yes	Yes	0	3	16	0	Yes	Sirs without n	Undifferentia	06.04.2010		Fungalinfectic	culture negat	yes
7.6	09.04.2010	.	.	3	Yes	Yes	Yes	Yes	0	4	44	0	Yes	Sepsis	Bacterial	21.04.2010	coag neg s	nosuspcion	culture negat	yes
7.05	14.04.2010	03.05.2010	.	3	Yes	Yes	Yes	Yes	0	4	21	0	Yes	Sepsis	Bacterial	19.04.2010	nfgnb	Fungalinfectic	culture negat	yes
7.6	14.04.2010	.	.	3	Yes	Yes	Yes	Yes	0	4	46	4	Yes	Sepsis	Bacterial	15.04.2010	nfgnb	Fungalinfectic	culture negat	no
5.6	14.04.2010	30.04.2010	29.04.2010	3	Yes	Yes	Yes	Yes	0	0	0	0	Yes	Sepsis	Bacterial	12.05.2010	coag neg s	Fungalinfectic	culture negat	yes
9	02.04.2010	08.05.2010	08.05.2010	3	Yes	Yes	Yes	Yes	0	3	8	0	Yes	Sepsis	Bacterial	28.04.2010	nfgnb	nosuspcion	culture negat	yes
11	26.04.2010	10.05.2010	10.05.2010	3	Yes	Yes	Yes	Yes	0	4	20	0	Yes	Sirs without n	Undifferentia	22.04.2010		Fungalinfectic	culture negat	yes
8.81	29.04.2010	.	.	3	Yes	Yes	Yes	Yes	0	0	0	0	Yes	Sepsis	Bacterial	29.04.2010	nfgnb	nosuspcion	culture negat	no
.	03.05.2010	.	.	0	Yes	Yes	Yes	Yes	0	2	16	15	Yes	Sepsis	Bacterial	26.04.2010	candida	Fungalinfectic	culture positi	yes
21	06.05.2010	20.05.2010	16.05.2010	3	Yes	Yes	Yes	Yes	0	1	2	0	Yes	Sepsis	Undifferentia	08.05.2010	nfgnb	nosuspcion	culture negat	yes
4.08	10.05.2010	28.05.2010	05.06.2010	3	Yes	Yes	Yes	Yes	0	5	42	2	Yes	Sirs without n	Undifferentia	04.05.2010		nosuspcion	culture negat	yes
22	12.05.2010	24.05.2010	24.05.2010	3	Yes	Yes	Yes	Yes	0	4	10	0	Yes	Sirs without n	Undifferentia	07.05.2010		nosuspcion	culture negat	yes
10	12.05.2010	26.05.2010	23.05.2010	3	Yes	Yes	Yes	Yes	0	0	0	0	Yes	Sirs without n	Undifferentia	18.05.2010		nosuspcion	culture negat	yes

14.3	14.05.2010	27.05.2010	12.06.2010	3	Yes	Yes	Yes	Yes	0	6	16	3	Yes	Sirs without n	Undifferentia	25.05.2010		Fungalinfectic	culture positi	no
32.8	19.05.2010	30.05.2010	19.05.2009	2	Yes	Yes	Yes	Yes	0	1	0	0	Yes	Sirs without n	Undifferentia	28.05.2010		nosuspcion	culture negat	yes
9.9	24.05.2010	12.06.2010	13.06.2010	3	Yes	Yes	Yes	Yes	0	4	24	0	Yes	Sirs without n	Undifferentia	01.06.2010		nosuspcion	culture negat	yes
9.4	28.05.2010	10.06.2010	11.06.2010	3	Yes	Yes	Yes	Yes	0	4	24	0	Yes	Sepsis	Undifferentia	06.06.2010	coag neg s	Fungalinfectic	culture negat	yes
10	31.05.2010	12.06.2010	31.05.2009	3	Yes	Yes	Yes	Yes	0	2	16	0	Yes	Sepsis	Undifferentia	27.05.2010	staph	nosuspcion	culture negat	no
2.6	08.06.2010	19.06.2010	16.06.2010	3	Yes	Yes	Yes	Yes	0	4	14	0	Yes	Sepsis	Undifferentia	02.07.2010	pseudomona	Fungalinfectic	culture negat	yes
8.8	07.06.2010	28.06.2010	.	3	Yes	Yes	Yes	Yes	0	14	126	8	Yes	Sepsis	Undifferentia	13.07.2010	kleb	Fungalinfectic	culture negat	yes
4.65	07.06.2010	23.06.2010	26.07.2010	3	Yes	Yes	Yes	Yes	0	8	91	8	Yes	Sirs without n	Undifferentia	07.06.2010		Fungalinfectic	culture negat	yes
9.9	12.06.2010	27.06.2010	26.06.2010	3	Yes	Yes	Yes	Yes	0	4	24	5	Yes	Sirs without n	Undifferentia	10.06.2010		Fungalinfectic	culture negat	yes
5.86	23.06.2010	08.07.2010	06.07.2010	3	Yes	Yes	Yes	Yes	0	4	20	0	Yes	Sirs without n	Undifferentia	22.06.2010		Fungalinfectic	culture positi	yes
21.7	23.06.2010	10.07.2010	03.07.2010	2	Yes	Yes	Yes	Yes	0	2	10	0	Yes	Sirs without n	Undifferentia	24.06.2010		nosuspcion	culture negat	yes
21	25.06.2010	09.07.2010	25.06.2010	3	Yes	Yes	Yes	Yes	0	0	0	0	Yes	Sirs without n	Undifferentia	29.06.2010		nosuspcion	culture negat	yes
21	28.06.2010	.	.	3	Yes	Yes	Yes	Yes	0	6	0	0	Yes	Sirs without n	Undifferentia	02.07.2010		nosuspcion	culture negat	yes
4.8	30.06.2010	.	.	3	Yes	Yes	Yes	Yes	0	3	26	0	Yes	Sepsis	Undifferentia	11.08.2010	coag neg s	nosuspcion	culture negat	yes
7	01.07.2010	17.07.2010	16.07.2010	3	Yes	Yes	Yes	Yes	0	3	20	3	Yes	Sepsis	Undifferentia	06.08.2010	kleb	Fungalinfectic	culture negat	yes
7.67	07.07.2010	21.07.2010	17.07.2010	3	Yes	Yes	Yes	Yes	0	2	18	0	Yes	Sirs without n	Undifferentia	12.07.2010		nosuspcion	culture negat	yes
22	17.07.2010	31.07.2010	30.07.2010	3	Yes	Yes	Yes	Yes	0	5	8	0	Yes	Sirs without n	Undifferentia	14.07.2010		nosuspcion	culture negat	yes
4.19	26.07.2010	08.08.2010	07.08.2010	3	Yes	Yes	Yes	Yes	0	2	11	0	Yes	Sirs without n	Undifferentia	13.07.2010		Fungalinfectic	culture negat	yes
24.5	26.07.2010	.	.	3	Yes	Yes	Yes	Yes	0	8	84	9	Yes	Sepsis	Bacterial	02.08.2010	staph	Fungalinfectic	culture negat	yes
14.6	28.07.2010	12.08.2010	23.08.2010	3	Yes	Yes	Yes	Yes	0	4	24	0	Yes	Sepsis	Undifferentia	04.08.2010	nfgnb	nosuspcion	culture negat	yes
6.48	31.07.2010	.	.	3	Yes	Yes	Yes	Yes	0	3	40	10	Yes	Sirs without n	Undifferentia	29.07.2010		Fungalinfectic	culture negat	yes
7.62	02.08.2010	17.08.2010	15.08.2010	2	Yes	Yes	Yes	Yes	0	2	8	0	Yes	Sirs without n	Undifferentia	11.08.2010		Fungalinfectic	culture negat	yes
14.8	04.08.2010	.	.	3	Yes	Yes	Yes	Yes	0	3	10	0	Yes	Sirs without n	Undifferentia	12.08.2010		nosuspcion	culture negat	yes
.	11.08.2010	.	.	3	Yes	Yes	Yes	Yes	0	2	14	0	Yes	Sepsis	Bacterial	10.08.2010	coag neg s	Fungalinfectic	culture negat	no
16.2	14.08.2010	28.08.2010	26.08.2010	3	Yes	Yes	Yes	Yes	0	2	6	0	Yes	Sirs without n	Undifferentia	12.08.2010		nosuspcion	culture negat	yes
10.4	23.08.2010	.	.	3	Yes	Yes	Yes	Yes	0	4	34	1	Yes	Sepsis	Bacterial	28.08.2010	pseudomona	nosuspcion	culture negat	yes
30.2	26.08.2010	06.09.2010	04.09.2010	3	Yes	Yes	Yes	Yes	0	8	11	1	Yes	Sepsis	Bacterial	01.10.2010	coag neg s	Fungalinfectic	culture negat	yes
9.3	30.08.2010	15.09.2010	04.10.2010	3	Yes	Yes	Yes	Yes	0	5	28	0	Yes	Sirs without n	Undifferentia	04.09.2010		nosuspcion	culture negat	yes
12.3	30.08.2010	10.09.2010	.	2	Yes	Yes	Yes	Yes	0	1	0	0	Yes	Sirs without n	Undifferentia	08.09.2010		nosuspcion	culture negat	yes
12.7	01.09.2010	15.09.2010	.	3	Yes	Yes	Yes	Yes	0	13	74	6	Yes	Sirs without n	Undifferentia	05.09.2010		Fungalinfectic	culture negat	yes
9.15	01.09.2010	13.09.2010	13.09.2010	3	Yes	Yes	Yes	Yes	0	1	10	0	Yes	Sirs without n	Undifferentia	09.09.2010		Fungalinfectic	culture negat	yes
7.59	13.09.2010	.	.	3	Yes	Yes	Yes	Yes	0	14	63	6	Yes	Sirs without n	Undifferentia	14.09.2010		Fungalinfectic	culture negat	yes
25.45	15.09.2010	.	.	3	Yes	Yes	Yes	Yes	0	10	0	0	Yes	Sepsis	Bacterial	12.09.2010	Ecoli	Fungalinfectic	culture negat	no
6	17.09.2010	02.10.2010	30.09.2010	3	Yes	Yes	Yes	Yes	0	6	26	0	Yes	Sirs without n	Undifferentia	13.09.2010		nosuspcion	culture negat	yes
9.6	18.09.2010	03.10.2010	01.10.2010	0	Yes	Yes	Yes	Yes	0	7	0	0	Yes	Sepsis	Bacterial	23.09.2010	coag neg s	Fungalinfectic	culture negat	yes

2.05	23.09.2010	08.10.2010	.	3	Yes	Yes	Yes	Yes	0	6	36	14	Yes	Sepsis	Bacterial	08.10.2010	pseudomona	Fungalinfectic	culture positi	yes
8.7	23.09.2010	09.10.2010	06.10.2010	3	Yes	Yes	Yes	Yes	0	5	28	0	Yes	Sepsis	Bacterial	18.09.2010	Ecoli	Fungalinfectic	culture negat	yes
5.77	25.09.2010	10.10.2010	10.10.2010	3	Yes	Yes	Yes	Yes	0	11	18	0	Yes	Sirs without n	Undifferentia	30.09.2010		Fungalinfectic	culture negat	yes
5.37	29.09.2010	14.10.2010	11.10.2010	3	Yes	Yes	Yes	Yes	0	2	2	4	Yes	Sirs without n	Undifferentia	07.10.2010		nosuspcion	culture negat	yes
8.84	09.10.2010	.	.	3	Yes	Yes	Yes	Yes	0	1	10	0	Yes	Sirs without n	Undifferentia	16.10.2010		Fungalinfectic	culture negat	yes
7.97	11.10.2010	.	.	3	Yes	Yes	Yes	Yes	0	3	26	0	Yes	Sepsis	Bacterial	20.11.2010	Acitenobac	Fungalinfectic	culture negat	yes
17.04	14.10.2010	01.11.2010	28.11.2010	3	Yes	Yes	Yes	Yes	0	5	18	0	Yes	Sirs without n	Undifferentia	14.10.2011		nosuspcion	culture negat	yes
12.47	18.10.2010	21.10.2010	31.10.2010	3	Yes	Yes	Yes	Yes	0	5	30	0	Yes	Sirs without n	Undifferentia	19.11.2010		Fungalinfectic	culture negat	yes
37.7	19.10.2010	.	.	2	Yes	Yes	Yes	Yes	0	1	0	0	Yes	Sirs without n	Undifferentia	28.10.2010		nosuspcion	culture negat	yes
5.84	20.10.2010	.	.	2	Yes	Yes	Yes	Yes	0	5	66	0	Yes	Sirs without n	Undifferentia	20.10.2010		Fungalinfectic	culture negat	yes
8.13	21.10.2010	.	.	3	Yes	Yes	Yes	Yes	0	4	18	0	Yes	Sirs without n	Undifferentia	29.10.2010		Fungalinfectic	culture negat	yes
5.54	28.10.2010	.	.	2	Yes	Yes	Yes	Yes	0	1	4	0	Yes	Sirs without n	Undifferentia	06.11.2010		nosuspcion	culture negat	yes
6.1	12.11.2010	.	.	2	Yes	Yes	Yes	Yes	0	4	18	0	Yes	Sepsis	Bacterial	17.11.2010	coag neg s	nosuspcion	culture negat	yes
18.2	15.11.2010	28.11.2010	25.11.2010	3	Yes	Yes	Yes	Yes	0	1	16	0	Yes	Sirs without n	Undifferentia	12.11.2010		nosuspcion	culture negat	yes
5.48	15.11.2010	.	.	2	Yes	Yes	Yes	Yes	0	3	24	0	Yes	Sirs without n	Undifferentia	25.11.2010		nosuspcion	culture negat	yes
13	30.11.2010	16.12.2010	13.12.2010	3	Yes	Yes	Yes	Yes	0	2	6	0	Yes	Sirs without n	Undifferentia	07.12.2010		nosuspcion	culture negat	yes
11.3	01.12.2010	15.12.2010	16.12.2010	3	Yes	Yes	Yes	Yes	0	4	28	0	Yes	Sirs without n	Undifferentia	01.12.2010		nosuspcion	culture negat	no
6.4	03.12.2010	.	.	3	Yes	Yes	Yes	Yes	0	0	0	0	Yes	Sepsis	Bacterial	27.12.2010	kleb	Fungalinfectic	culture negat	yes
7.3	03.12.2010	.	.	2	Yes	Yes	Yes	Yes	0	3	20	0	Yes	Sepsis	Bacterial	28.11.2010	pseudomona	Fungalinfectic	culture negat	yes
2.66	04.12.2010	.	.	3	Yes	Yes	Yes	Yes	0	7	8	0	Yes	Sirs without n	Undifferentia	11.12.2010		nosuspcion	culture negat	yes
2.74	06.12.2010	20.12.2010	22.12.2010	3	Yes	Yes	Yes	Yes	0	7	97	2	Yes	Sirs without n	Undifferentia	25.12.2010		Fungalinfectic	culture negat	no
7.24	10.12.2010	.	.	0	Yes	Yes	Yes	Yes	0	9	58	0	Yes	Sepsis	Bacterial	08.12.2010	enterobact	Fungalinfectic	culture negat	yes
11.8	17.12.2010	.	.	3	Yes	Yes	Yes	Yes	0	6	47	0	Yes	Sepsis	Bacterial	21.12.2010	coag neg s	Fungalinfectic	culture negat	no
4.2	24.12.2010	.	.	0	NO	NO	NO	NO	0	2	14	0	Yes	Sirs without n	Undifferentia	17.12.2010		nosuspcion	culture negat	no
8.17	29.12.2010	15.01.2011	24.01.2011	3	Yes	Yes	Yes	Yes	0	4	30	0	Yes	Sirs without n	Undifferentia	04.01.2011		nosuspcion	culture negat	yes
6.4	29.12.2010	13.01.2011	15.01.2011	3	Yes	Yes	Yes	Yes	0	8	66	2	Yes	Sepsis	Bacterial	04.02.2011	nfgnb	Fungalinfectic	culture negat	yes
14.6	30.12.2010	12.01.2011	12.01.2011	3	Yes	Yes	Yes	Yes	0	5	10	0	Yes	Sirs without n	Undifferentia	06.01.2011		Fungalinfectic	culture negat	yes
11.1	06.01.2011	.	.	.	.	.	.	.	0	4	24	0	Yes	Sepsis	Bacterial	10.01.2011	coag neg s	Fungalinfectic	culture negat	yes
14.1	11.01.2011	.	.	.	.	.	.	.	0	7	33	0	Yes	Sirs without n	.	16.01.2011		Fungalinfectic	.	yes
12.2	12.01.2011	.	.	.	.	.	.	.	0	7	20	0	Yes	Sepsis	Bacterial	26.01.2011	pseudo	Fungalinfectic	culture negat	yes
4.4	13.01.2011	27.01.2011	25.01.2011	.	.	.	.	.	0	2	6	0	Yes	Sirs without n	.	14.02.2011		.	.	yes
6.99	13.01.2011	.	.	.	.	.	.	.	0	18	1	0	Yes	Sepsis	Fungal	18.01.2011	candida	Fungalinfectic	culture positi	yes
2.14	20.01.2011	.	.	.	0	0	0	0	0	7	34	3	Yes	Sirs without n	.	25.01.2011		.	.	no
20.7	24.01.2011	07.02.2011	03.02.2011	.	.	.	.	.	0	7	12	0	Yes	Sirs without n	.	27.01.2011		Fungalinfectic	culture negat	yes
7.4	28.01.2011	12.02.2011	12.02.2011	.	.	.	.	.	0	15	70	0	Yes	Sepsis	Fungal	15.04.2011	candida	Fungalinfectic	culture positi	yes

7.89	02.02.2011	19.02.2011	25.02.2011	.	.	.	.	.	0	7	44	1	Yes	Sirs without n	.	07.02.2011		Fungalinfectic	culture negat	yes
6.29	03.02.2011	17.02.2011	16.02.2011	.	.	.	.	.	0	1	8	0	Yes	Sirs without n	.	09.02.2011		.	.	yes
5.38	05.02.2011	20.02.2011	16.02.2011	.	.	.	.	.	0	4	16	0	Yes	Sirs without n	.	11.02.2011		Fungalinfectic	culture negat	yes
9.87	09.12.2011	28.02.2011	28.02.2011	.	.	.	.	.	0	5	14	0	Yes	Sirs without n	.	04.02.2011		.	.	yes
4.03	10.02.2011	23.02.2011	24.02.2011	.	.	.	.	.	0	6	66	8	Yes	Sepsis	Bacterial	26.05.2011	pseudo	nosuspcion	.	yes
10.55	11.02.2011	24.02.2011	21.02.2011	.	.	.	.	.	0	2	14	0	Yes	Sirs without n	.	02.02.2011		.	.	yes
7.8	17.02.2011	.	.	.	.	.	.	.	0	1	10	2	Yes	Sirs without n	.	23.02.2011		Fungalinfectic	culture negat	yes
7.87	18.02.2011	.	.	0	0	0	0	0	0	4	22	0	Yes	Sepsis	Bacterial	28.02.2011	Ecoli	Fungalinfectic	.	yes
27.6	22.02.2011	.	.	.	.	.	.	.	0	5	22	0	Yes	Sirs without n	.	16.02.2011		Fungalinfectic	culture negat	no
5.6	22.02.2012	12.03.2012	.	.	.	.	.	.	0	28	168	42	Yes	Sepsis	Bacterial	14.04.2011	nfgnb	Fungalinfectic	culture negat	no
12.6	02.03.2011	.	.	3	Yes	Yes	Yes	Yes	0	5	20	0	Yes	Sirs without n	.	26.02.2011		nosuspcion	.	yes
23.9	03.03.2011	.	.	.	.	.	.	.	0	1	18	0	Yes	Sirs without n	.	09.03.2011		.	.	yes
7.4	03.03.2011	16.03.2011	17.03.2011	.	.	.	.	.	0	1	2	0	Yes	Sirs without n	.	11.03.2011		nosuspcion	.	yes
11.8	10.03.2011	23.03.2011	21.03.2011	.	.	.	.	.	0	4	12	0	Yes	Sepsis	.	12.05.2011	coag neg s	Fungalinfectic	culture negat	yes
12.2	14.03.2011	31.03.2011	.	.	.	.	.	.	0	5	18	0	Yes	Sepsis	Bacterial	12.03.2011	a,strep	Fungalinfectic	culture negat	yes
4.74	16.03.2011	28.03.2011	01.04.2011	.	.	.	.	.	0	20	34	0	Yes	Sirs without n	.	17.03.2011		Fungalinfectic	culture negat	yes
11.6	17.03.2011	29.03.2011	30.03.2011	.	.	.	.	.	0	3	16	0	Yes	.	.	15.03.2011		.	.	no
12.1	24.03.2011	.	.	.	.	.	.	.	0	6	137	0	Yes	Sepsis	Bacterial	03.04.2011	kleb	nosuspcion	.	yes
11.5	24.03.2011	09.04.2011	09.04.2011	.	.	.	.	.	0	6	54	1	Yes	Sirs without n	.	31.03.2011		nosuspcion	.	yes
11.1	25.03.2011	.	.	.	.	.	.	.	0	14	94	0	Yes	.	.	.		.	.	no
6.12	30.03.2011	15.04.2011	16.04.2011	.	.	.	.	.	0	2	4	0	Yes	Sepsis	Bacterial	24.03.2012	coag neg s	nosuspcion	.	yes
17.2	30.03.2011	.	.	3	NO	NO	NO	NO	0	4	14	0	Yes	Sepsis	Bacterial	29.05.2011	E coli	Fungalinfectic	culture negat	yes
19	31.03.2011	15.04.2011	16.04.2011	3	Yes	Yes	Yes	Yes	0	3	8	0	Yes	.	.	23.03.2011		.	.	yes
11	11.04.2011	26.04.2011	23.04.2011	0	Yes	Yes	Yes	Yes	0	4	11	0	Yes	.	.	16.04.2011		.	.	yes
12.7	14.04.2011	.	.	.	.	.	.	.	0	0	6	0	Yes	.	.	.		.	.	no
12.9	25.04.2011	.	.	3	NO	NO	NO	NO	0	5	26	0	Yes	Sirs without n	Bacterial	11.05.2011	nfgnb	Fungalinfectic	culture negat	yes
8.4	25.04.2011	06.05.2011	07.05.2011	0	Yes	Yes	Yes	Yes	0	5	22	0	Yes	Sepsis	Bacterial	27.04.2011	coag neg s	Fungalinfectic	culture negat	yes
5.16	28.04.2011	.	.	.	.	.	.	.	0	23	108	5	Yes	.	.	.		Fungalinfectic	culture negat	yes
11.8	02.05.2011	19.05.2011	16.05.2011	3	Yes	.	.	.	0	1	14	0	Yes	Sirs without n	.	13.05.2011		.	.	yes
11.3	02.05.2011	13.05.2011	.	.	.	.	.	.	0	2	8	0	Yes	Sirs without n	.	01.05.2011		Fungalinfectic	culture negat	yes
8.2	17.05.2011	28.05.2011	31.05.2011	.	.	.	.	.	0	3	16	0	Yes	Sirs without n	.	25.05.2011		nosuspcion	.	yes
9.49	18.05.2011	02.06.2011	31.05.2011	.	.	.	.	.	0	0	4	0	Yes	.	.	.		.	.	no
24.6	23.05.2011	02.06.2011	04.06.2011	.	.	.	.	.	0	4	26	0	Yes	.	.	14.06.2011		Fungalinfectic	culture negat	yes
7.4	23.05.2011	.	.	.	.	.	.	.	0	2	6	0	Yes	.	.	.		.	.	no
13.3	25.05.2011	09.06.2011	09.06.2011	.	.	.	.	.	0	4	14	0	Yes	Sirs without n	.	28.05.2011		.	.	yes

12.71	09.06.2011	23.06.2011	29.06.2011	.	.	.	.	.	0	6	14	0	Yes	Sirs without n	.		10.06.2011		nosuspcion	.	yes
12.2	10.06.2011	04.07.2011	21.06.2011	.	.	.	.	.	0	1	4	0	Yes	Sirs without n	.		10.06.2011		nosuspcion	.	yes
12.7	10.06.2011	25.06.2011	23.06.2011	.	.	.	.	.	0	1	10	0	Yes	Sirs without n	.		22.06.2011		Fungalinfectic	culture negat	yes
12.49	13.06.2011	04.07.2011	27.07.2011	.	.	.	.	.	0	8	14	0	Yes	Sepsis	Bacterial		20.06.2011	pseudo	nosuspcion	.	yes
15.19	15.06.2012	01.07.2011	01.07.2011	.	.	.	.	.	0	12	4	0	Yes	Sepsis	Bacterial		28.06.2011	Kleb	nosuspcion	.	yes
14.8	15.06.2011	02.07.2011	29.06.2011	.	.	.	.	.	0	1	0	0	Yes	Sepsis	Bacterial		29.08.2011	pseudo	nosuspcion	.	yes
31.06	23.06.2011	05.07.2011	14.07.2011	.	.	.	.	.	0	14	102	0	Yes	Sepsis	Bacterial		24.09.2011	enterococc	Fungalinfectic	culture positi	yes
16.8	29.06.2011	.	.	.	.	.	.	.	0	0	8	0	Yes	Sepsis	Bacterial		23.07.2012	kleb	Fungalinfectic	.	yes
15.9	07.07.2011	.	.	.	.	.	.	.	0	2	20	0	Yes	Sepsis	Bacterial		14.09.2011	NFGNB	Fungalinfectic	culture negat	yes
15.96	07.07.2011	21.07.2011	18.07.2011	.	.	.	.	.	0	3	4	0	Yes	Sirs without n	.		.		Fungalinfectic	culture negat	yes
11.3	11.07.2011	27.07.2011	04.08.2011	.	.	.	.	.	0	4	20	0	Yes	Sirs without n	.		19.07.2011		nosuspcion	.	yes
23.6	11.07.2011	.	.	.	.	.	.	.	0	15	14	12	Yes	Sepsis	Bacterial		10.07.2011	pseudo	Fungalinfectic	culture negat	yes
4.6	13.07.2011	28.07.2011	27.07.2011	.	.	.	.	.	0	3	14	0	Yes	Sirs without n	.		15.07.2011		nosuspcion	.	yes
24.4	21.07.2011	31.07.2011	30.07.2011	.	.	.	.	.	0	4	18	0	Yes	Sepsis	Bacterial		26.07.2011	kleb	nosuspcion	.	yes
19.4	06.10.2011	02.08.2011	31.07.2011	.	.	.	.	.	0	0	8	0	Yes	Sirs without n	.		.		nosuspcion	.	yes
6.48	29.07.2011	15.08.2011	11.08.2011	.	.	.	.	.	0	1	14	0	Yes	Sepsis	Bacterial		04.08.2011	coagnestap	nosuspcion	.	yes
4.04	01.08.2011	18.08.2011	19.08.2011	.	.	.	.	.	0	5	20	0	Yes	Sepsis	Bacterial		08.08.2011	strep	nosuspcion	.	yes
13.1	03.08.2011	18.08.2011	27.08.2011	.	.	.	.	.	0	2	25	0	Yes	Sepsis	Bacterial		08.08.2011	staph	nosuspcion	.	yes
22.6	22.07.2011	18.08.2011	17.08.2011	.	.	.	.	.	0	2	7	0	Yes	Sirs without n	.		09.08.2011		Fungalinfectic	culture negat	yes
17.5	06.08.2011	17.08.2011	17.08.2011	.	.	.	.	.	0	4	14	0	Yes	Sepsis	Bacterial		06.08.2011	coagnestap	Fungalinfectic	culture negat	yes
6.5	16.08.2011	.	.	.	.	.	.	.	0	0	1	0	Yes	Sepsis	Bacterial		18.08.2011	coagnestap	Fungalinfectic	culture negat	no
16.3	13.08.2011	26.08.2011	28.08.2011	.	.	.	.	.	0	15	78	0	Yes	Sepsis	Bacterial		11.09.2011	coagnestap	Fungalinfectic	culture negat	yes
12.7	13.08.2011	27.08.2011	29.08.2011	.	.	.	.	.	0	0	0	0	Yes	Sirs without n	.		22.08.2011		.	.	yes
13.7	17.08.2011	01.09.2011	01.09.2011	.	.	.	.	.	0	0	0	0	Yes	Sirs without n	.		.		nosuspcion	.	yes
.	22.08.2011	.	.	.	.	.	.	.	0	3	28	0	Yes	Sepsis	Bacterial		26.09.2011	coagnestap	Fungalinfectic	culture negat	yes
5.1	27.08.2011	.	.	.	.	.	.	.	0	0	0	0	Yes	Sirs without n	.		27.08.2011		Fungalinfectic	.	yes
4.2	31.08.2011	.	.	.	.	.	.	.	0	4	8	0	Yes	Sirs without n	.		05.09.2011		nosuspcion	.	yes
11.6	18.09.2011	21.09.2011	20.09.2011	.	.	.	.	.	0	4	13	0	Yes	Sepsis	Bacterial		04.09.2011	ECOLI	nosuspcion	.	yes
12.9	08.09.2011	.	.	.	.	.	.	.	0	13	88	0	Yes	Sepsis	Bacterial		23.09.2011	enterococc	Fungalinfectic	culture negat	yes
22.4	08.09.2011	20.09.2011	21.09.2011	.	.	.	.	.	0	11	26	0	Yes	Sepsis	Bacterial		03.11.2011	kleb	Fungalinfectic	culture negat	yes
8.14	10.09.2011	26.09.2011	21.09.2011	.	.	.	.	.	0	4	18	0	Yes	Sirs without n	.		08.09.2011		Fungalinfectic	culture negat	yes
13.9	14.09.2011	29.09.2011	29.09.2011	.	.	.	.	.	0	5	2	0	Yes	Sepsis	Bacterial		21.09.2011	kleb	nosuspcion	.	yes
14.9	16.09.2011	29.09.2011	28.09.2011	.	.	.	.	.	0	16	58	28	Yes	Sepsis	Bacterial		28.10.2011	kleb	Fungalinfectic	culture negat	yes
10	21.09.2011	.	.	.	.	.	.	.	0	0	0	0	Yes	.	.		.		.	.	no
1.01	22.09.2011	.	.	.	.	.	.	.	0	1	22	0	Yes	Sirs without n	.		.		nosuspcion	.	yes

10	23.09.2011	.	.	.	.	.	.	.	0	6	57	0	Yes	Sirs without n	.	23.09.2011		Fungalinfectic	culture negat	no
24.1	27.09.2011	11.10.2011	.	.	.	.	.	.	0	0	0	0	Yes	Sepsis	Bacterial	03.11.2011	coagnegsta	nosuspcion	.	yes
16.1	28.09.2011	13.10.2011	09.10.2011	.	.	.	.	.	0	2	4	0	Yes	Sepsis	Bacterial	04.10.2011	strep	.	.	yes
11.2	12.10.2011	26.10.2011	24.10.2011	.	.	.	.	.	0	15	14	10	Yes	Sirs without n	.	20.10.2011		.	.	yes
7.9	17.10.2011	.	.	.	.	.	.	.	0	2	16	0	Yes	Sepsis	Bacterial	27.10.2011	KLEB	nosuspcion	.	yes
3.9	17.10.2011	06.11.2011	13.11.2011	.	.	.	.	.	0	5	38	0	Yes	Sirs without n	.	12.10.2011		Fungalinfectic	culture negat	yes
10.1	19.10.2011	15.11.2011	02.11.2011	.	.	.	.	.	0	2	8	0	Yes	Sirs without n	.	25.10.2011		nosuspcion	.	yes
11	20.10.2011	04.11.2011	.	.	.	.	.	.	0	1	2	0	Yes	Sepsis	Bacterial	24.10.2011	coagnegsta	Fungalinfectic	culture negat	no
10.4	25.10.2011	10.11.2011	05.11.2011	.	.	.	.	.	0	0	0	0	Yes	Sepsis	Bacterial	16.11.2011	coagnegsta	Fungalinfectic	.	yes
6.1	27.10.2011	09.11.2011	13.11.2011	.	.	.	.	.	0	4	17	0	Yes	.	.	.		Fungalinfectic	culture negat	yes
9	01.11.2011	.	.	.	.	.	.	.	0	3	7	0	Yes	Sepsis	Bacterial	06.11.2011	coagnegsta	.	.	yes
24	09.11.2011	.	.	.	.	.	.	.	0	4	24	0	Yes	Sirs without n	.	14.11.2011		.	.	yes
5.9	12.11.2011	28.11.2011	25.11.2011	.	.	.	.	.	0	2	6	0	Yes	Sirs without n	.	.		Fungalinfectic	.	yes
9.39	16.11.2011	.	.	.	.	.	.	.	0	4	2	0	Yes	Sirs without n	.	21.11.2011		.	.	yes
10	17.11.2011	03.12.2011	30.11.2011	.	.	.	.	.	0	6	15	0	Yes	Sirs without n	.	19.11.2011		.	.	yes
20.1	18.11.2011	.	.	.	.	.	.	.	0	0	0	0	NO	.	.	.		.	.	no
4.01	21.11.2011	07.12.2011	05.12.2011	.	.	.	.	.	0	11	32	0	Yes	Sepsis	Bacterial	27.11.2011	nfgnb	Fungalinfectic	culture negat	yes
1.99	23.11.2011	11.12.2011	.	.	.	.	.	.	0	21	175	5	Yes	Sirs without n	.	.		.	.	yes
11	24.11.2011	10.12.2011	06.12.2011	.	.	.	.	.	0	3	4	4	Yes	.	.	.		.	.	yes
9.98	03.12.2011	16.12.2011	13.12.2011	.	.	.	.	.	0	3	14	0	Yes	Sirs without n	.	06.12.2011		nosuspcion	.	yes
12.8	12.12.2011	25.12.2011	24.12.2011	.	.	.	.	.	0	1	4	0	Yes	Sepsis	Bacterial	13.04.2011	enterococc	Fungalinfectic	culture positi	yes
8.29	13.12.2011	29.12.2011	23.12.2011	.	.	.	.	.	0	4	12	0	Yes	Sepsis	Bacterial	12.12.2011	coagnestap	Fungalinfectic	culture negat	yes
10.09	15.12.2011	29.12.2011	30.12.2011	.	.	.	.	.	0	4	31	6	Yes	Sepsis	Bacterial	24.11.2012	Haemphilus	Fungalinfectic	culture negat	yes
9.25	15.12.2011	01.01.2012	06.01.2012	.	.	.	.	.	0	9	52	20	Yes	Sepsis	.	15.12.2011		Fungalinfectic	culture negat	yes
13	21.12.2011	15.01.2012	16.01.2012	.	.	.	.	.	0	5	40	0	Yes	Sirs without n	.	22.12.2011		nosuspcion	.	yes
14.7	29.12.2011	14.01.2011	28.03.2011	.	.	.	.	.	0	19	223	0	Yes	Sirs without n	.	05.01.2011		Fungalinfectic	culture negat	no
2.27	30.12.2011	.	.	.	.	.	.	.	0	4	24	0	Yes	Sepsis	Bacterial	07.01.2011	nfgnb	Fungalinfectic	culture negat	yes
1.33	16.06.2010	.	.	0	NO	NO	NO	NO	0	3	29	8	Yes	Sepsis	Bacterial	22.06.2010	coag neg s	Fungalinfectic	culture negat	no
10	15.03.2010	11.04.2010	.	3	Yes	Yes	Yes	Yes	0	6	30	0	Yes	Sirs without n	Undifferentia	08.03.2010		Fungalinfectic	culture negat	no

amikacindt	duratio	Ampl	DT Ampho	Durat	Colisti	DTColistin	Duratio	SOS	SOSdt	Muco	Mucositisdt	GVHD	GVHDdt	VZV	VZV	HSV	HSV	CMV	CMVdt	BKV	BKVdt	CSA
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
14.01.2010	1	yes	15.01.2010	30	yes	16.01.10	19	NO	.	no	.	yes	22.02.2010	No	.	0	.	Yes	08.02.2010		0	.	Yes
20.01.2010	1	no	.	.	no	.	.	NO	.	no	.	yes	22.02.2010	No	.	0	.	0	.		0	.	Yes
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
.	.	yes	27.01.2010	13	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
.	.	yes	26.01.2010	13	no	.	.	NO	.	yes	26.01.2010	yes	09.03.2010	No	.	0	.	Yes	11.03.2010		0	.	Yes
14.02.2010	3	yes	25.01.2010	17	yes	21.01.10	15	YES	14.02.2010	no	.	no	.	No	.	0	.	Yes	03.03.2010		0	.	Yes
08.02.2010	1	yes	09.02.2010	5	yes	10.02.10	5	NO	.	no	.	yes	23.02.2010	No	.	0	.	0	.		0	.	Yes
16.02.2010	3	yes	21.02.2010	6	yes	25.02.10	5	NO	.	yes	17.02.2010	no	.	No	.	0	.	0	.		0	.	Yes
.	.	yes	24.02.2010	5	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
19.02.2010	2	yes	21.02.2010	11	no	.	.	NO	.	yes	22.02.2010	yes	28.02.2010	No	.	0	.	0	.		0	.	Yes
16.02.2010	2	yes	21.02.2010	19	no	.	.	NO	.	yes	20.02.2010	yes	10.03.2010	No	.	0	.	Yes	04.03.2010		0	.	Yes
26.02.2010	1	no	.	.	no	.	.	NO	.	yes	16.03.2010	no	.	No	.	0	.	0	.		0	.	Yes
.	.	yes	24.02.2010	3	no	.	.	NO	.	yes	08.03.2010	no	.	No	.	0	.	0	.		0	.	Yes
24.02.2010	16	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
14.03.2010	11	no	.	.	no	.	.	NO	.	no	.	yes	27.03.2010	No	.	0	.	0	.		0	.	Yes
15.03.2010	5	yes	21.03.2010	4	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
27.03.2010	1	yes	28.03.2010	3	no	.	.	NO	.	yes	28.03.2010	yes	05.05.2010	No	.	0	.	0	.		0	.	Yes
27.03.2010	1	no	.	.	no	.	.	NO	.	yes	26.03.2010	yes	25.04.2010	No	.	0	.	Yes	18.04.2010		0	.	Yes
.	.	yes	08.04.2009	7	no	.	.	NO	.	no	.	yes	20.04.2010	No	.	0	.	Yes	26.04.2010		0	.	Yes
30.03.2010	2	yes	31.03.2010	17	no	.	.	NO	.	no	.	yes	05.05.2010	No	.	0	.	Yes	28.05.2010		0	.	NO
05.04.2010	2	yes	08.04.2010	6	no	.	.	NO	.	no	.	yes	16.05.2010	No	.	0	.	0	.		0	.	Yes
07.04.2010	11	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
12.04.2010	20	yes	19.04.2010	14	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
.	.	yes	19.04.2010	6	yes	21.04.10	4	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
12.04.2010	3	yes	16.04.2010	12	no	.	.	NO	.	yes	26.04.2010	yes	15.05.2010	No	.	0	.	Yes	25.05.2010	Yes		15.06.2005	Yes
01.05.2010	8	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
23.04.2010	3	yes	07.05.2010	4	yes	07.05.10	5	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
.	.	no	.	.	no	.	.	NO	.	no	.	yes	17.05.2010	No	.	0	.	Yes	26.05.2010		0	.	Yes
20.04.2010	4	yes	28.04.2010	8	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	NO
08.05.2010	11	no	.	.	yes	19.05.10	2	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
15.05.2010	11	no	.	.	no	.	.	NO	.	yes	16.05.2010	yes	31.05.2010	No	.	0	.	0	.		0	.	Yes
16.05.2010	9	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
18.05.2010	2	no	.	.	no	.	.	NO	.	no	.	yes	30.05.2010	No	.	0	.	0	.		0	.	Yes

.	.	yes	30.05.2010	13	yes	25.05.10	3	NO	.	no	.	yes	27.05.2010	No	.	0	.	Yes	02.06.2010		0	.	Yes
28.05.2010	1	no	.	.	no	.	.	NO	.	yes	26.06.2009	yes	23.06.2010	No	.	0	.	0	.		0	.	NO
03.05.2010	10	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	21.06.2010		0	.	Yes
27.05.2010	16	no	.	.	yes	09.06.10	3	NO	.	no	.	no	.	No	.	0	.	Yes	10.06.2010		0	.	Yes
.	.	no	.	.	yes	06.06.10	8	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
04.06.2010	4	yes	06.06.2010	12	yes	04.07.10	4	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
11.06.2010	7	no	.	.	no	.	.	YES	14.06.2010	no	.	no	.	No	.	0	.	Yes	30.06.2010		0	.	Yes
06.06.2010	24	no	.	.	no	.	.	YES	29.06.2010	yes	29.06.2010	no	.	No	.	0	.	0	.		0	.	NO
08.06.2010	8	yes	15.06.2010	19	yes	18.06.10	5	NO	.	yes	22.06.2010	yes	15.07.2010	No	.	0	.	Yes	15.07.2010		0	.	Yes
27.06.2010	2	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	16.08.2010	Yes		05.07.2010	Yes
24.06.2010	15	no	.	.	no	.	.	NO	.	yes	28.06.2010	no	.	No	.	0	.	Yes	26.07.2010		0	.	NO
29.06.2010	11	no	.	.	no	.	.	NO	.	yes	10.07.2010	no	.	No	.	0	.	0	.		0	.	NO
05.07.2010	5	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
05.07.2010	2	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	26.07.2010		0	.	Yes
04.07.2010	2	yes	05.07.2010	15	no	.	.	NO	.	no	.	yes	02.08.2010	No	.	0	.	0	.	Yes		26.08.2010	Yes
11.07.2010	10	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
14.07.2010	7	no	.	.	no	.	.	NO	.	yes	18.07.2010	no	.	No	.	0	.	0	.		0	.	Yes
22.07.2010	3	yes	04.08.2010	9	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
02.08.2010	1	yes	04.08.2010	5	no	.	.	NO	.	no	.	yes	15.09.2010	No	.	0	.	0	.		0	.	Yes
03.08.2010	11	no	.	.	no	.	.	YES	09.08.2010	no	.	no	.	No	.	0	.	0	.		0	.	Yes
01.08.2010	1	yes	10.08.2010	6	no	.	.	NO	.	no	.	yes	20.09.2010	No	.	0	.	0	.		0	.	Yes
07.08.2010	1	yes	17.08.2010	10	no	.	.	NO	.	no	.	yes	27.09.2010	No	.	0	.	Yes	30.08.2010	Yes		29.11.2010	NO
12.08.2010	11	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	Yes
.	.	yes	04.08.2010	10	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	NO
11.08.2010	2	no	.	.	no	.	.	NO	.	yes	15.08.2010	yes	12.08.2010	No	.	0	.	0	.		0	.	Yes
28.08.2010	4	no	.	.	no	.	.	YES	29.08.2010	no	.	no	.	No	.	0	.	0	.		0	.	Yes
16.09.2010	6	yes	01.09.2010	18	yes	02.10.10	11	NO	.	no	.	yes	09.09.2010	No	.	0	.	0	.		0	.	Yes
03.09.2010	8	no	.	.	no	.	.	NO	.	no	.	yes	30.09.2010	No	.	0	.	Yes	18.10.2010		0	.	Yes
08.09.2010	4	no	.	.	no	.	.	NO	.	yes	08.09.2010	no	.	No	.	0	.	0	.		0	.	NO
05.09.2010	6	yes	12.09.2010	13	no	.	.	YES	09.09.2010	yes	07.09.2010	no	.	No	.	0	.	0	.		0	.	Yes
08.09.2010	2	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	20.09.2010		0	.	Yes
13.09.2010	5	yes	25.09.2010	14	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	12.10.2010		0	.	Yes
.	.	yes	11.09.2010	10	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.	Yes		16.09.2010	.
15.09.2010	6	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	08.11.2010		0	.	Yes
20.09.2010	3	yes	27.09.2010	6	yes	01.10.10	5	NO	.	no	.	no	.	No	.	0	.	0	.		0	.	.

22.09.2010	2	yes	03.10.2010	8	yes	25.09.10	13	YES	29.09.2010	no	.	no	.	No	.	0	.	0	.		0	.		NO
18.09.2010	2	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	24.10.2010		0	.		Yes
01.10.2010	2	yes	03.10.2010	14	yes	06.10.10	3	NO	.	yes	01.10.2010	yes	14.10.2010	No	.	0	.	0	.		0	.		Yes
06.10.2010	6	no	.	.	no	.	.	NO	.	yes	04.10.2010	yes	15.11.2010	No	.	0	.	0	.		0	.		Yes
16.10.2010	7	yes	16.10.2010	4	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		Yes
19.10.2010	2	yes	20.10.2011	5	no	.	.	NO	.	no	.	yes	12.11.2010	No	.	0	.	Yes	18.11.2010		0	.		Yes
14.10.2011	4	no	.	.	no	.	.	NO	.	no	.	yes	21.10.2010	No	.	0	.	0	.		0	.		Yes
19.11.2010	2	yes	22.10.2010	10	no	.	.	NO	.	no	.	yes	04.11.2010	No	.	0	.	0	.		0	.		Yes
05.11.2010	5	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		NO
28.10.2010	12	no	.	.	no	.	.	NO	.	yes	27.10.2010	yes	16.11.2010	No	.	0	.	Yes	05.12.2010		0	.		NO
28.10.2010	5	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		Yes
05.11.2010	5	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		NO
17.11.2010	2	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		NO
15.11.2010	7	no	.	.	no	.	.	NO	.	yes	22.11.2010	yes	20.12.2010	No	.	0	.	Yes	08.10.2010		0	.		Yes
25.11.2010	8	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		NO
06.12.2010	6	no	.	.	no	.	.	NO	.	no	.	yes	21.01.2011	No	.	0	.	0	.		0	.		Yes
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		Yes
30.11.2010	3	yes	03.12.2010	11	no	.	.	NO	.	yes	05.12.2010	yes	20.02.2011	No	.	0	.	Yes	20.01.2011		0	.		Yes
08.12.2010	2	yes	08.12.2010	4	no	.	.	NO	.	yes	13.12.2010	yes	20.12.2010	No	.	0	.	Yes	09.12.2010		0	.		Yes
10.12.2010	2	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		Yes
.	.	yes	15.12.2010	18	no	.	.	YES	11.12.2010	yes	10.12.2010	no	.	No	.	0	.	Yes	30.12.2010		0	.		Yes
04.12.2010	11	yes	04.12.2010	18	yes	29.12.10	12	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		.
.	.	yes	19.12.2010	14	yes	30.12.10	2	NO	.	no	.	no	.	No	.	0	.	Yes	13.01.2011		0	.		Yes
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		NO
04.01.2011	8	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		Yes
01.01.2011	5	yes	19.12.2010	43	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	10.02.2011		0	.		Yes
05.01.2011	7	no	.	.	no	.	.	NO	.	no	.	yes	24.01.2011	No	.	0	.	0	.		0	.		Yes
10.01.2011	3	yes	05.01.2011	17	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		Yes
16.01.2011	3	yes	31.01.2011	4	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		Yes
13.01.2011	3	yes	18.01.2011	10	yes	22.01.11	7	NO	.	no	.	no	.	No	.	0	.	Yes	21.01.2011		0	.		Yes
14.01.2011	7	no	.	.	no	.	.	NO	.	no	.	yes	29.01.2011	No	.	0	.	0	.	Yes		06.05.2011		Yes
19.01.2011	3	yes	20.01.2011	6	yes	20.01.11	2	NO	.	no	.	no	.	No	.	0	.	0	.		0	.		Yes
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	04.03.2011		0	.		Yes
27.01.2011	4	yes	06.02.2011	9	no	.	.	NO	.	no	.	yes	.	No	.	0	.	0	.		0	.		Yes
02.02.2011	1	yes	06.02.2011	9	no	.	.	NO	.	yes	11.02.2011	yes	.	No	.	0	.	Yes	03.03.2011		0	.		.

07.02.2011	15	yes	11.02.2011	16	no	.	.	NO	.	no	.	yes	19.04.2011	No	.	0	.	0	.			0	.	Yes
08.02.2011	3	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.			0	.	Yes
11.02.2011	5	yes	21.02.2011	1	no	.	.	NO	.	yes	14.02.2012	yes	05.05.2011	No	.	0	.	0	.			0	.	NO
04.02.2011	3	no	.	.	no	.	.	NO	.	yes	.	no	.	No	.	0	.	0	.			0	.	Yes
13.05.2011	4	no	.	.	yes	28.05.11	.	NO	.	yes	18.02.2011	no	.	No	.	0	.	0	.			0	.	NO
06.02.2011	5	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.			0	.	Yes
23.02.2011	2	no	.	.	yes	25.02.11	7	NO	.	yes	27.02.2011	yes	03.03.2011	No	.	0	.	0	.			0	.	Yes
19.02.2011	17	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.	Yes	26.03.2011			NO
.	.	yes	08.03.2011	3	no	.	.	NO	.	no	.	yes	.	No	.	0	.	Yes	07.04.2011			0	.	Yes
.	.	yes	23.02.2011	1	yes	09.03.11	3	NO	.	yes	23.02.2011	yes	04.03.2011	No	.	0	.	Yes	17.03.2011	Yes		.		Yes
27.02.2011	11	no	.	.	no	.	.	NO	.	no	.	no	.	chick	14.(	0	.	0	.			0	.	Yes
08.03.2011	4	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	05.04.2011			0	.	Yes
11.03.2011	6	no	.	.	no	.	.	NO	.	yes	10.03.2011	yes	19.03.2011	No	.	0	.	Yes	18.04.2011			0	.	Yes
15.03.2011	2	yes	17.03.2011	6	no	.	.	NO	.	yes	12.03.2011	yes	28.03.2011	No	.	0	.	Yes	07.04.2011			0	.	Yes
25.03.2011	2	yes	28.03.2011	3	no	.	.	YES	30.03.2011	no	.	no	.	No	.	0	.	0	.			0	.	Yes
16.03.2011	7	yes	22.03.2011	2	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	23.04.2011			0	.	Yes
.	.	no	.	.	no	.	.	NO	.	yes	26.03.2011	yes	01.04.2011	No	.	Yes	.	Yes	02.05.2011	Yes		11.04.2011		NO
23.03.2011	16	no	.	.	yes	18.03.11	23	NO	.	no	.	no	.	No	.	0	.	0	.			0	.	Yes
30.03.2011	5	no	.	.	no	.	.	NO	.	no	.	yes	25.04.2012	No	.	0	.	NO	.			0	.	Yes
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.			0	.	Yes
04.04.2011	5	no	.	.	no	.	.	NO	.	no	.	yes	25.04.2011	No	.	0	.	Yes	05.05.2011			0	.	Yes
28.03.2011	2	yes	18.04.2011	9	yes	09.04.11	18	NO	.	no	.	yes	11.05.2011	No	.	0	.	Yes	19.05.2011			0	.	NO
31.03.2011	12	no	.	.	no	.	.	NO	.	yes	.	yes	.	No	.	0	.	0	.			0	.	Yes
15.04.2011	5	no	.	.	no	.	.	NO	.	yes	.	no	.	No	.	0	.	0	.			0	.	NO
.	.	no	.	.	no	.	.	NO	.	yes	18.04.2011	no	.	No	.	0	.	0	.			0	.	Yes
28.04.2011	2	yes	30.04.2011	19	yes	09.05.11	9	NO	.	yes	.	no	.	No	.	0	.	Yes	20.06.2011			0	.	NO
27.04.2011	2	yes	30.04.2011	18	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.			0	.	Yes
06.05.2011	3	yes	07.11.2011	6	no	.	.	NO	.	no	.	yes	19.09.2011	No	.	0	.	0	.			0	.	NO
08.05.2011	5	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	27.05.2011			0	.	NO
10.05.2011	2	yes	13.05.2011	5	yes	13.05.11	12	NO	.	yes	.	yes	24.05.2011	No	.	0	.	Yes	21.11.2011			0	.	NO
25.05.2011	6	no	.	.	no	.	.	NO	.	yes	26.05.2011	no	.	No	.	0	.	Yes	13.02.2011			0	.	NO
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.			0	.	Yes
14.06.2011	3	yes	20.05.2011	19	yes	28.05.11	9	NO	.	yes	.	no	.	No	.	0	.	0	.			0	.	Yes
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.			0	.	NO
28.05.2011	7	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.			0	.	Yes

12.06.2011	5	no	.	.	no	.	.	NO	.		no	.		yes	02.07.2011	No	.	0	.	Yes	21.07.2011		0	.	Yes
10.06.2011	7	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
17.06.2011	6	yes	29.06.2011	3	no	.	.	NO	.		no	.		no	.	No	.	0	.	Yes	25.08.2011		0	.	Yes
20.06.2011	4	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
28.06.2011	8	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
30.06.2011	5	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	Yes	16.08.2011		0	.	NO
24.06.2011	5	yes	17.07.2011	2	yes	13.07.11	8	NO	.		no	.		yes	12.07.2011	No	.	0	.	Yes	04.08.2011		0	.	Yes
08.07.2011	11	no	.	.	yes	10.07.11	9	NO	.		no	.		no	.	No	.	0	.	Yes	21.07.2011		0	.	NO
07.07.2011	1	yes	07.07.2011	8	no	.	.	NO	.		no	.		no	.	No	.	0	.	Yes	15.06.2011		0	.	Yes
12.07.2011	2	yes	17.07.2011	6	no	.	.	NO	.		no	.		yes	27.07.2011	No	.	0	.	Yes	12.08.2011		0	.	Yes
19.07.2011	7	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	Yes	22.08.2011		0	.	Yes
19.07.2011	8	yes	01.08.2011	9	no	.	.	NO	.		no	.		yes	28.08.2011	No	.	0	.	Yes	16.08.2011		0	.	Yes
15.07.2011	11	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
14.07.2011	4	no	.	.	yes	28.07.11	4	NO	.		yes	19.04.2011		no	.	No	.	0	.	0	.		0	.	Yes
24.07.2011	2	no	.	.	yes	30.07.11	15	NO	.		no	.		yes	05.08.2011	No	.	0	.	Yes	06.10.2011		0	.	Yes
04.08.2011	1	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	Yes	06.09.2011		0	.	Yes
07.08.2011	6	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
08.08.2011	2	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
09.08.2011	3	yes	11.08.2011	3	no	.	.	NO	.		yes	03.08.2011		no	.	No	.	0	.	Yes	22.08.2011		0	.	Yes
05.08.2011	3	yes	12.08.2011	3	no	.	.	NO	.		no	.		yes	.	No	.	0	.	0	.		0	.	Yes
.	.	yes	18.08.2011	7	no	.	.	NO	.		no	.		no	.	No	.	0	.	Yes	25.11.2011		0	.	.
01.09.2011	5	yes	19.08.2011	8	yes	21.08.11	16	NO	.		no	.		yes	25.08.2011	No	.	0	.	Yes	07.10.2011		0	.	Yes
22.08.2011	7	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
28.08.2011	3	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
26.08.2011	2	yes	20.08.2011	1	yes	29.08.11	10	NO	.		no	.		no	.	No	.	0	.	Yes	22.08.2011		0	.	NO
27.08.2011	2	no	.	.	yes	05.09.11	2	YES	31.08.2011		yes	31.08.2011		no	.	No	.	0	.	0	.		0	.	Yes
05.09.2011	9	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
04.09.2011	1	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
05.09.2011	1	yes	17.09.2011	15	no	.	.	NO	.		yes	17.09.2011		no	.	No	.	0	.	0	.		0	.	Yes
10.09.2011	3	yes	16.09.2011	8	no	.	.	NO	.		yes	14.09.2011		no	.	No	.	0	.	Yes	27.10.2011		0	.	Yes
08.09.2011	3	yes	10.09.2011	7	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
20.09.2011	2	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	Yes	31.10.2011		0	.	Yes
23.09.2011	7	yes	23.09.2011	7	yes	30.10.11	4	NO	.		yes	.		yes	21.09.2011	No	.	0	.	Yes	07.11.2011		0	.	Yes
.	.	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	0	.		0	.	Yes
02.10.2011	6	no	.	.	no	.	.	NO	.		no	.		no	.	No	.	0	.	Yes	26.01.2012		0	.	Yes

.	.	no	.	.	yes	09.10.11	7	NO	.	no	.	no	.	No	.	0	.	0	.	0	.	Yes
08.10.2011	3	no	.	.	no	.	.	NO	.	yes	10.10.2011	no	.	No	.	0	.	0	.	0	.	NO
04.10.2011	6	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.	0	.	Yes
20.10.2011	11	no	.	.	no	.	.	NO	.	yes	15.10.2011	yes	.	No	.	0	.	Yes	06.12.2011	0	.	Yes
07.10.2011	8	no	.	.	yes	28.10.11	4	NO	.	yes	.	no	.	No	.	0	.	0	.	0	.	Yes
12.10.2011	13	yes	01.11.2011	6	no	.	.	NO	.	yes	21.10.2011	no	.	No	.	0	.	0	.	0	.	Yes
25.10.2011	1	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	01.12.2011	0	.	Yes
.	.	yes	30.10.2011	7	no	.	.	NO	.	yes	26.10.2011	no	.	No	.	0	.	Yes	16.11.2011	0	.	Yes
25.10.2011	11	no	.	.	no	.	.	NO	.	no	.	yes	.	No	.	0	.	Yes	04.11.2011	0	.	Yes
30.10.2011	6	yes	06.11.2011	4	yes	06.11.11	5	NO	.	no	.	no	.	No	.	0	.	0	.	0	.	Yes
12.11.2011	7	yes	19.12.2011	27	no	.	.	NO	.	yes	11.11.2011	no	.	No	.	0	.	NO	.	0	.	NO
13.11.2011	13	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	16.12.2011	0	.	Yes
17.11.2011	7	yes	17.11.2011	5	no	.	.	NO	.	yes	.	no	.	No	.	0	.	0	.	0	.	Yes
21.11.2011	1	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.	0	.	Yes
19.11.2011	6	no	.	.	no	.	.	YES	09.11.2011	yes	.	no	.	No	.	0	.	Yes	19.12.2011	0	.	Yes
.	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.	0	.	Yes
27.11.2011	6	yes	29.11.2011	4	no	.	.	NO	.	yes	07.12.2011	yes	06.02.2011	No	.	0	.	Yes	27.02.2011	0	.	NO
23.11.2011	11	yes	05.12.2011	54	yes	06.02.11	7	YES	30.11.2011	no	.	yes	.	No	.	0	.	Yes	03.12.2011	0	.	Yes
16.02.2011	5	yes	17.02.2012	11	yes	22.02.12	3	NO	.	yes	11.02.2012	yes	.	No	.	0	.	0	.	0	.	.
27.11.2011	4	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	24.01.2012	0	.	Yes
01.04.2011	7	yes	19.03.2011	27	no	.	.	NO	.	yes	19.12.2011	no	.	No	.	0	.	0	.	0	.	Yes
12.12.2011	13	yes	01.02.2012	19	no	.	.	NO	.	no	.	no	.	No	.	0	.	Yes	16.01.2012	0	.	Yes
20.12.2011	4	yes	14.01.2012	15	no	.	.	NO	.	yes	.	yes	19.01.2012	No	.	0	.	Yes	27.01.2012	0	.	Yes
12.12.2011	13	yes	16.12.2011	9	yes	06.01.12	.	NO	.	no	.	yes	31.12.2011	No	.	0	.	0	.	0	.	NO
22.12.2011	.	no	.	.	no	.	.	NO	.	no	.	no	.	No	.	0	.	0	.	0	.	Yes
.	.	yes	05.01.2011	3	yes	12.01.11	12	YES	02.01.2011	yes	.	no	.	No	.	0	.	0	.	0	.	Yes
01.01.2011	5	yes	05.01.2011	16	yes	10.01.11	4	NO	.	no	.	yes	13.01.2011	No	.	0	.	Yes	16.01.2011	0	.	Yes
.	.	yes	10.06.2010	9	yes	22.06.10	6	NO	.	no	.	no	.	No	.	0	.	Yes	21.06.2010	0	.	NO
.	.	yes	01.04.2010	18	no	.	.	YES	05.04.2010	no	.	yes	03.04.2010	No	.	0	.	Yes	01.04.2010	0	.	Yes

CSAc01	CSA1c0DT	CSA2c0	CSA2c0DT	AKIRIFLE	AKIAKIN	AKIyes/no	AKI stage	NumberAKI	DtAKI	RIFLE1	DtAKI	AKIN1	cause	DtAKI
160	04.01.2010	1246	08.01.2010	NOAKI	CLASS1	AKI	AKI STAGE 1	1	.	.	01.03.10	CLASS1	NEPHROTOXI	.
90	08.11.2010	100	12.01.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	22.02.10	RISK	22.01.10	CLASS1	GUT GVHD/A	.
120	15.01.2010	105	22.01.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	15.03.10	RISK	15.03.10	CLASS1	IDIOPATHIC	.
250	26.01.2010	405	26.01.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
30	16.01.2010	57	19.01.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
320	19.01.2010	298	27.01.2010	RISK	CLASS1	AKI	AKI STAGE 1	3	31.01.10	RISK	31.01.10	CLASS1	SIRS	15.02.10
100	28.01.2010	130	02.02.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	03.03.10	RISK	03.03.10	CLASS1	SIRS	03.03.10
40	02.02.2010	365	05.02.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
200	10.02.2010	90	12.02.2010	RISK	CLASS1	AKI	AKI STAGE 1	3	19.02.10	RISK	19.02.10	CLASS1	SIRS	22.02.10
360	11.02.2010	105	16.02.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	03.03.10	RISK	03.03.10	CLASS1	IDIOPATHIC	.
450	13.02.2010	230	19.02.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	04.03.10	RISK	04.03.10	CLASS1	GUT GVHD/A	09.03.10
200	18.02.2010	92	23.02.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	31.03.10	RISK	31.03.10	CLASS1	CO	.
90	18.02.2010	50	23.02.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	19.03.10	RISK	19.03.10	CLASS1	IDIOPATHIC	.
170	24.02.2010	103	26.02.2010	INJURY	CLASS2	AKI	.	1	13.03.10	INJURY	13.03.10	CLASS2	IDIOPATHIC	.
330	25.02.2010	220	02.03.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	07.03.10	RISK	07.03.10	CLASS1	NEPHROTOXI	.
190	09.03.2010	182	16.03.2010	FAILURE	CLASS3	AKI	.	1	27.03.10	FAILURE	27.03.10	CLASS3	CO	.
140	11.03.2010	135	14.03.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	08.04.10	RISK	08.04.10	CLASS1	IDIOPATHIC	.
280	16.03.2010	410	19.03.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	12.04.10	RISK	30.03.10	CLASS1	CO	.
70	19.03.2010	103	23.03.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
290	30.03.2010	111	03.04.2010	RISK	CLASS1	AKI	AKI STAGE 1	3	02.04.10	RISK	02.04.10	CLASS1	SIRS	11.04.10
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	1	10.04.10	RISK	10.04.10	CLASS1	SIRS	.
240	31.03.2010	178	03.04.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	14.04.10	RISK	14.04.10	CLASS1	CO	.
270	08.04.2010	470	13.04.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	03.05.10	RISK	03.05.10	CLASS1	CO	.
60	10.04.2010	285	16.04.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
200	12.04.2010	64	16.04.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	21.04.10	RISK	21.04.10	CLASS1	SIRS	.
200	14.04.2010	149	16.04.2010	INJURY	CLASS2	AKI	.	1	22.04.10	INJURY	22.04.10	CLASS2	SIRS	.
80	18.04.2010	125	21.04.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
180	25.04.2010	350	27.04.2010	INJURY	CLASS2	AKI	.	1	08.05.10	INJURY	08.05.10	CLASS2	HYPOTENSIOI	.
150	28.04.2010	210	30.04.2010	INJURY	CLASS2	AKI	.	1	17.06.10	INJURY	17.06.10	CLASS2	SIRS	.
.	.	.	.	FAILURE	CLASS3	AKI	.	1	01.05.10	FAILURE	01.05.10	CLASS3	SIRS	.
60	05.05.2010	75	07.05.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
120	04.05.2010	240	11.05.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	02.06.10	RISK	02.06.10	CLASS1	SIRS	.
240	11.05.2010	113	14.05.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	02.06.10	RISK	02.06.10	CLASS1	CO	.
340	10.05.2010	288	14.05.2010	FAILURE	CLASS3	AKI	.	1	03.06.10	FAILURE	03.06.10	CLASS3	GUT GVHD/A	.

300	12.05.2010	460	21.05.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	23.05.10	RISK	21.05.10	CLASS1	HYPOTENSIOI	12.06.10
.	.	.	.	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
100	21.05.2010	60	25.05.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
300	22.05.2010	333	01.06.2010	INJURY	CLASS2	AKI	.	2	14.06.10	INJURY	14.06.10	CLASS2	SIRS	14.06.10
60	28.05.2010	110	08.06.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	21.06.10	RISK	21.06.10	CLASS1	CO	.
140	02.06.2010	250	08.06.2010	FAILURE	CLASS3	AKI	.	2	07.06.10	INJURY	07.06.10	CLASS2	NEPHROTOXI	28.06.10
195	04.06.2010	167	08.06.2010	FAILURE	CLASS3	AKI	.	1	21.06.10	FAILURE	21.06.10	CLASS3	SIRS	.
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	1	12.07.10	RISK	12.07.10	CLASS1	SIRS	.
240	11.06.2010	90	18.06.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	18.06.10	RISK	18.06.10	CLASS1	SIRS	05.07.10
200	21.06.2010	200	25.06.2010	INJURY	CLASS2	AKI	.	1	11.07.10	INJURY	11.07.10	CLASS2	IDIOPATHIC	.
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	1	26.07.10	RISK	26.07.10	CLASS1	SIRS	.
.	.	.	.	INJURY	CLASS2	AKI	.	1	17.07.10	INJURY	17.07.10	CLASS2	SIRS	.
130	27.06.2010	105	29.06.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	05.08.10	RISK	05.08.10	CLASS1	CO	.
200	28.06.2010	175	02.07.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
300	30.06.2010	111	02.07.2010	RISK	CLASS1	AKI	AKI STAGE 1	3	15.07.10	RISK	15.07.10	CLASS1	SIRS	29.07.10
170	06.07.2010	65	09.07.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
170	16.07.2010	250	20.07.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	09.08.10	RISK	09.08.10	CLASS1	IDIOPATHIC	.
60	25.07.2010	420	27.07.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	16.08.10	RISK	16.08.10	CLASS1	IDIOPATHIC	.
210	25.07.2010	115	27.07.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	09.08.10	RISK	09.08.10	CLASS1	SIRS	.
100	24.07.2010	350	27.07.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
110	30.07.2010	95	03.08.2010	INJURY	CLASS2	AKI	.	2	10.08.10	RISK	10.08.10	CLASS1	SIRS	13.09.10
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	1	16.08.10	RISK	16.08.10	CLASS1	SIRS	.
40	04.08.2010	90	03.08.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
.	.	.	.	INJURY	CLASS2	AKI	.	1	08.08.10	INJURY	08.08.10	CLASS2	SIRS	.
140	13.08.2010	210	17.08.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	31.08.10	RISK	31.08.10	CLASS1	IDIOPATHIC	.
20	09.08.2010	305	24.08.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	30.08.10	RISK	30.08.10	CLASS1	SIRS	.
90	30.08.2010	240	31.08.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	11.09.10	RISK	11.09.10	CLASS1	CO	06.10.10
100	26.08.2010	110	31.08.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	1	16.09.10	RISK	16.09.10	CLASS1	SIRS	.
100	03.08.2009	205	03.09.2009	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
300	31.08.2010	370	04.09.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	09.09.10	RISK	09.09.10	CLASS1	SIRS	18.10.10
130	10.09.2010	108	14.09.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	08.10.10	RISK	08.10.10	CLASS1	NEPHROTOXI	.
.	.	.	.	FAILURE	CLASS3	AKI	.	1	17.09.10	FAILURE	17.09.10	CLASS3	SIRS	.
200	17.09.2010	240	18.09.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	14.10.10	RISK	21.09.10	CLASS1	SIRS	.
.	.	.	.	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.

.	.	.	.	FAILURE	CLASS3	AKI	.	1	07.10.10	FAILURE	07.10.10	CLASS3	SIRS	.
200	22.09.2010	147	24.09.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	11.10.10	RISK	11.10.10	CLASS1	CO	.
180	26.09.2010	255	28.09.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	05.10.10	RISK	05.10.10	CLASS1	GUT GVHD/A	25.10.10
190	27.09.2010	161	01.10.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
300	07.10.2010	848	12.10.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	17.10.10	RISK	17.10.10	CLASS1	NEPHROTOXI	.
200	09.10.2010	250	12.10.2010	FAILURE	CLASS3	AKI	.	2	21.10.10	RISK	21.10.10	CLASS1	CO	08.11.10
160	11.10.2010	77	15.10.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	13.12.10	RISK	13.12.10	CLASS1	IDIOPATHIC	.
300	17.10.2010	395	19.10.2010	INJURY	CLASS2	AKI	.	2	03.11.10	RISK	03.11.10	CLASS1	CO	14.11.10
.	.	.	.	INJURY	CLASS2	AKI	.	2	05.11.10	INJURY	05.11.10	CLASS2	SIRS	.
.	.	.	.	INJURY	CLASS2	AKI	.	2	17.11.10	INJURY	17.11.10	CLASS2	SIRS	.
150	20.10.2010	200	25.10.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
.	.	.	.	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	1	02.12.10	RISK	02.12.10	CLASS1	TAC TOXICITY	.
100	14.11.2010	110	16.11.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	06.12.10	RISK	06.12.10	CLASS1	CO	.
.	.	.	.	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
130	27.11.2010	311	03.12.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	20.01.10	RISK	20.01.11	CLASS1	CO	.
150	23.11.2010	130	30.11.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
250	01.12.2010	130	03.12.2010	INJURY	CLASS2	AKI	.	2	12.12.10	INJURY	12.12.10	CLASS2	SIRS	30.12.10
300	16.12.2010	433	31.12.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
300	02.12.2010	235	07.12.2010	RISK	CLASS1	AKI	AKI STAGE 1	2	11.12.10	RISK	11.12.10	CLASS1	SIRS	23.12.10
120	02.12.2010	430	07.12.2010	INJURY	CLASS2	AKI	.	2	27.12.10	RISK	27.12.10	CLASS1	CO	11.01.11
.	.	.	.	INJURY	CLASS2	AKI	.	1	18.12.10	INJURY	18.12.10	CLASS2	SIRS	18.12.10
200	15.12.2010	285	21.12.2010	FAILURE	CLASS3	AKI	.	1	30.12.10	FAILURE	30.12.10	CLASS3	SIRS	.
.	.	.	.	INJURY	CLASS2	AKI	.	1	18.12.10	INJURY	18.12.10	CLASS2	SIRS	.
80	25.12.2010	128	28.12.2010	NOAKI	NOAKI	NO AKI	NO AKI	0	.	.	.	.	.	.
160	28.12.2010	126	31.12.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	03.01.11	RISK	03.01.11	CLASS1	TAC TOXICITY	.
350	29.12.2010	285	31.12.2010	RISK	CLASS1	AKI	AKI STAGE 1	1	26.12.10	RISK	26.12.10	CLASS1	CO	.
100	05.01.2011	153	07.01.2011	RISK	CLASS1	AKI	AKI STAGE 1	1	11.03.11	RISK	11.03.11	CLASS1	SIRS	.
70	08.01.2011	102	11.01.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
100	09.01.2011	65	14.01.2011	FAILURE	CLASS3	AKI	.	1	27.01.11	FAILURE	27.01.11	CLASS3	SIRS	.
350	12.01.2011	210	17.01.2011	RISK	CLASS1	AKI	AKI STAGE 1	2	04.02.11	RISK	02.02.11	CLASS1	SIRS	07.03.11
50	11.01.2011	210	14.01.2011	FAILURE	CLASS3	AKI	.	1	23.01.11	FAILURE	23.01.11	CLASS3	SIRS	.
300	19.01.2011	210	21.01.2011	INJURY	CLASS2	AKI	.	2	25.02.11	INJURY	25.02.11	CLASS2	SIRS	31.01.11
200	21.01.2011	230	25.01.2011	INJURY	CLASS2	AKI	.	1	14.02.11	INJURY	12.02.11	CLASS2	SIRS	.
.	.	.	.	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.

100	30.01.2011	125	01.02.2011	INJURY	CLASS2	AKI	.	1	11.07.11	INJURY	11.07.11	CLASS2	IDIOPATHIC	.
400	02.02.2011	190	04.02.2011	RISK	CLASS1	AKI	AKI STAGE 1	2	17.02.11	RISK	15.02.11	CLASS1	SIRS	28.05.11
.	.	.	.	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
60	05.02.2011	65	08.02.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
.	.	.	.	NOAKI	CLASS1	AKI	AKI STAGE 1	1	.	.	27.02.11	CLASS1	CO	.
240	10.02.2011	350	15.02.2011	RISK	CLASS2	AKI	.	1	28.02.11	INJURY	28.02.11	CLASS2	CO	.
180	16.02.2011	250	19.02.2011	NOAKI	CLASS1	AKI	AKI STAGE 1	1	.	.	27.05.11	CLASS1	GUT GVHD/A	.
.	.	.	.	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
70	21.02.2011	110	25.02.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
170	22.02.2011	225	25.02.2011	FAILURE	CLASS3	AKI	.	2	08.03.11	INJURY	08.03.12	CLASS2	SIRS	18.04.11
80	26.02.2011	135	01.03.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
250	28.02.2011	285	18.03.2011	INJURY	CLASS2	AKI	.	1	24.03.11	INJURY	24.03.11	CLASS2	CO	.
320	01.03.2011	325	04.03.2011	FAILURE	CLASS3	AKI	.	2	02.04.11	INJURY	02.04.11	CLASS2	CO	11.07.11
290	08.03.2011	480	15.03.2011	INJURY	CLASS2	AKI	.	2	21.03.11	INJURY	21.03.11	CLASS2	CO	07.04.11
60	10.03.2011	230	15.03.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
300	15.03.2011	275	18.03.2011	RISK	CLASS1	AKI	AKI STAGE 1	3	24.03.11	RISK	24.03.11	CLASS1	NEPHROTOXI	11.04.11
.	.	.	.	NOAKI	CLASS1	AKI	.	.	.	.	.	.	.	.
60	21.03.2011	22	26.03.2011	FAILURE	CLASS3	AKI	.	1	09.04.11	FAILURE	09.04.11	CLASS3	SIRS	.
100	21.03.2011	117	25.03.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
150	25.03.2011	165	29.03.2011	INJURY	CLASS2	AKI	.	3	22.03.11	RISK	22.03.11	CLASS1	SIRS	04.05.11
80	26.03.2011	92	29.03.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	1	.	RISK	02.05.11	CLASS1	NEPHROTOXI	.
280	30.03.2011	180	01.04.2011	NOAKI	CLASS1	AKI	.	.	.	.	.	.	.	.
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	1	22.04.11	RISK	22.04.11	CLASS1	SIRS	.
100	13.04.2011	465	15.04.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
.	.	.	.	FAILURE	CLASS3	AKI	.	1	24.05.11	FAILURE	24.05.11	CLASS3	SIRS	.
400	23.04.2011	200	26.04.2011	INJURY	CLASS2	AKI	.	1	14.05.11	INJURY	14.05.11	CLASS2	CO	.
.	.	.	.	INJURY	CLASS2	AKI	.	3	14.05.11	INJURY	14.05.11	CLASS2	NEPHROTOXI	09.06.11
.	.	.	.	NOAKI	CLASS1	AKI	AKI STAGE 1	1	.	.	27.05.11	CLASS1	GUT GVHD/A	.
.	.	.	.	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.
.	.	.	.	RISK	CLASS1	AKI	AKI STAGE 1	2	13.06.11	RISK	13.06.11	CLASS1	CO	09.02.11
300	16.05.2011	390	20.05.2011	NOAKI	CLASS1	AKI	AKI STAGE 1	2	.	.	03.06.11	CLASS1	CO	30.06.11
120	23.05.2011	160	24.05.2011	INJURY	CLASS2	AKI	.	2	26.05.11	RISK	26.05.11	CLASS1	SIRS	18.07.11
.	.	.	.	INJURY	CLASS3	AKI	.	1	21.05.11	INJURY	21.05.11	CLASS3	SIRS	.
180	22.05.2011	230	24.05.2011	INJURY	CLASS2	AKI	.	1	11.07.11	INJURY	11.07.11	CLASS2	CO	.

200	05.06.2011	320	10.06.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
180	09.06.2011	250	14.06.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
170	17.06.2011	170	11.06.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
100	10.06.2011	150	14.06.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
80	12.06.2011	235	14.06.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
.	.	.	.	INJURY	CLASS2	AKI	.		1	01.09.11	INJURY	01.09.11	CLASS2	SIRS	.
260	22.06.2011	95	24.06.2011	INJURY	CLASS2	AKI	.		2	02.07.11	INJURY	02.07.11	CLASS2	SIRS	12.09.11
.	.	.	.	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
280	06.07.2011	160	08.07.2011	INJURY	CLASS2	AKI	.		1	28.07.11	INJURY	28.07.11	CLASS2	SIRS	.
140	06.07.2011	160	08.07.2011	INJURY	CLASS2	AKI	.		2	23.07.11	RISK	23.07.11	CLASS1	SIRS	07.09.11
60	07.07.2011	80	12.07.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
50	11.07.2011	200	15.07.2011	INJURY	CLASS2	AKI	.		1	10.07.11	INJURY	10.07.11	CLASS2	CO	.
120	10.07.2011	105	12.07.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
120	20.07.2011	80	26.07.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
100	20.07.2011	75	22.07.2011	NOAKI	CLASS1	AKI	AKI STAGE 1		1	.	.	17.10.11	CLASS1	IDIOPATHIC	.
280	28.07.2011	280	02.08.2011	INJURY	CLASS2	AKI	.		1	22.08.11	INJURY	22.08.11	CLASS2	CO	.
70	27.07.2011	60	02.08.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
90	31.07.2011	145	05.08.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
250	03.08.2011	200	05.08.2011	RISK	CLASS1	AKI	AKI STAGE 1		2	14.08.11	RISK	14.08.11	CLASS1	CO	27.02.11
220	05.08.2011	125	09.08.2011	FAILURE	CLASS3	AKI	.		2	.	.	01.08.11	CLASS1	SIRS	19.08.11
.	.	.	.	FAILURE	CLASS3	AKI	.		1	24.11.11	FAILURE	24.11.11	CLASS3	SIRS	.
50	12.08.2011	60	19.08.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
380	10.08.2011	225	16.08.2011	NOAKI	CLASS1	AKI	AKI STAGE 1		1	.	.	05.09.11	CLASS1	SIRS	.
350	15.08.2011	330	19.08.2011	NOAKI	CLASS1	AKI	AKI STAGE 1		1	.	.	28.09.11	CLASS1	CO	.
.	.	.	.	FAILURE	CLASS3	AKI	.		2	17.08.11	INJURY	17.08.11	CLASS2	SIRS	27.08.11
300	26.08.2011	75	01.09.2011	FAILURE	CLASS3	AKI	.		1	06.09.11	FAILURE	06.09.11	CLASS3	SIRS	.
60	27.08.2011	45	29.08.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	16.06.11	RISK	16.06.12	CLASS1	CO	.
230	07.09.2011	160	13.09.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	22.09.11	RISK	22.09.11	CLASS1	CO	.
100	18.09.2011	50	20.09.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
310	06.09.2011	260	09.09.2011	INJURY	CLASS2	AKI	.		3	03.09.11	RISK	03.09.11	CLASS1	SIRS	20.09.11
310	09.09.2011	140	06.09.2011	INJURY	CLASS2	AKI	.		2	23.09.11	INJURY	23.09.11	CLASS2	CO	21.06.11
150	11.09.2011	70	13.09.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	28.11.11	RISK	28.11.11	CLASS1	IDIOPATHIC	.
310	13.09.2011	160	13.09.2011	INJURY	CLASS2	AKI	.		2	29.09.11	INJURY	29.09.11	CLASS2	NEPHROTOXI	06.11.11
70	18.09.2011	50	20.09.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.	
360	21.09.2011	270	23.09.2011	INJURY	CLASS2	AKI	.		2	20.10.11	INJURY	20.10.11	CLASS2	SIRS	16.01.11

200	19.09.2011	190	27.09.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.		
.	.	.	.	INJURY	CLASS2	AKI	.		2	.	.	09.10.11	CLASS1	CO	15.10.11	
100	25.09.2011	96	27.09.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	07.11.11	RISK	07.11.11	CLASS1	SIRS	.	
400	12.10.2011	330	14.10.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	05.12.11	RISK	05.12.11	CLASS1	IDIOPATHIC	.	
235	18.10.2011	340	15.10.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.		
70	13.10.2011	100	18.10.2011	INJURY	CLASS2	AKI	.		1	28.03.11	INJURY	28.03.12	CLASS2	SIRS	.	
100	16.10.2011	100	21.10.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.		
100	19.10.2011	120	21.10.2011	RISK	CLASS1	AKI	AKI STAGE 1		2	01.12.11	RISK	01.12.11	CLASS1	SIRS	22.12.11	
300	24.10.2011	235	24.10.2011	INJURY	CLASS2	AKI	.		3	14.11.11	INJURY	14.11.11	CLASS2	SIRS	04.12.11	
220	24.10.2011	135	28.10.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	19.11.11	RISK	19.11.11	CLASS1	CO	.	
.	.	.	.	INJURY	CLASS2	AKI	.		3	16.01.12	RISK	16.01.12	CLASS1	NEPHROTOXI	07.05.11	
100	06.11.2011	110	08.11.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	19.01.12	RISK	19.01.12	CLASS2	.	.	
220	10.11.2011	125	15.11.2011	RISK	CLASS1	AKI	AKI STAGE 1		3	11.11.11	RISK	11.11.11	CLASS1	NEPHROTOXI	18.11.11	
100	13.11.2011	85	15.11.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	09.04.11	RISK	09.04.12	CLASS1	IDIOPATHIC	.	
200	14.11.2011	145	22.11.2011	RISK	CLASS1	AKI	AKI STAGE 1		2	16.11.11	RISK	16.11.11	CLASS1	SIRS	30.11.11	
240	16.11.2011	240	22.11.2011	RISK	CLASS1	AKI	AKI STAGE 1		1	02.12.11	RISK	02.12.11	CLASS1	CO	.	
.	.	.	.	INJURY	CLASS2	AKI	.		4	17.11.11	RISK	17.11.11	CLASS1	SIRS	01.12.11	
120	20.11.2011	200	22.11.2011	RISK	CLASS1	AKI	.		2	16.12.11	RISK	16.12.11	CLASS1	SIRS	31.12.11	
.	.	.	.	INJURY	CLASS2	AKI	.		2	12.12.11	INJURY	12.12.11	CLASS2	IDIOPATHIC	26.12.11	
380	02.12.2011	65	06.12.2011	INJURY	CLASS2	AKI	.		2	21.01.11	RISK	21.01.12	CLASS1	CO	23.02.11	
100	20.03.2011	100	03.04.2011	INJURY	CLASS2	AKI	.		2	01.01.12	RISK	01.01.12	CLASS1	TAC TOXICITY	15.01.12	
200	01.02.2012	300	07.02.2012	INJURY	CLASS2	AKI	.		3	26.12.11	INJURY	26.12.11	CLASS2	SIRS	18.02.11	
100	12.12.2011	65	16.12.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.		
.	.	.	.	INJURY	CLASS2	AKI	.		2	12.12.11	RISK	12.12.11	CLASS1	SIRS	22.12.11	
100	18.12.2011	125	23.12.2011	NOAKI	NOAKI	NO AKI	.	.	.	.	.	.	.	.		
250	26.12.2011	180	30.12.2011	INJURY	CLASS2	AKI	.		3	09.01.12	RISK	09.01.12	CLASS1	NEPHROTOXI	10.02.12	
350	29.12.2011	290	03.01.2011	INJURY	CLASS2	AKI	.		2	14.01.11	INJURY	14.01.12	CLASS2	NEPHROTOXI	23.01.11	
.	.	.	.	INJURY	CLASS2	AKI	.		1	24.06.10	INJURY	24.06.10	CLASS2	SIRS	.	
24	12.03.2010	147	17.03.2010	FAILURE	CLASS3	AKI	.		1	16.04.10	FAILURE	16.04.10	CLASS3	SIRS	.	

RIFLE2	DtAKI	AKIN2	cause	DtAKI	DtAKI	RIFLE3	AKIN3	cause	outcome	UTI	UTIdt	HD	HDdt
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
RISK	15.02.10	CLASS1	SIRS	09.03.10	09.03.10	RISK	CLASS1	IDIOPATHIC	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
RISK	22.02.10	CLASS1	NEPHROTOXI	04.03.10	04.03.10	RISK	CLASS1	SIRS	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
RISK	09.03.10	CLASS1	CO	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
RISK	11.04.10	CLASS1	SIRS	28.04.10	28.04.10	RISK	CLASS1	GVHD/AGE	recovered	no	08.04.2010	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	yes	14.06.2010	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.

RISK	12.06.10	CLASS1	SIRS	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.	
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.	
INJURY	28.06.10	CLASS1	SIRS	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	recovered	yes	26.05.2010	no	.	
FAILURE	28.06.10	CLASS3	SIRS	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.	
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.	
RISK	05.07.10	CLASS1	IDIOPATHIC	.	.	.	.	.	persistant rer	no	.	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	yes	24.08.2010	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.	
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.	
RISK	29.07.10	CLASS1	CO	07.08.10	07.08.10	RISK	CLASS1	SIRS	recovered	no	26.08.2010	no	.	
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.	
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.	
INJURY	13.09.10	CLASS2	CO	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	recovered	no	30.07.2010	no	.	
.	.	.	.	.	.	.	.	.	normal renal	yes	11.08.2010	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.	
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	yes	10.08.2010	no	.	
RISK	06.10.10	CLASS1	NEPHROTOXI	.	.	.	.	.	persistant rer	yes	01.10.2010	no	.	
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.	
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.	
RISK	18.10.10	CLASS1	CO	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	persistant rer	no	.	yes	18.09.2010	
.	14.10.10	CLASS1	CO	.	.	.	.	.	recovered	no	.	no	.	
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.	

.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
RISK	25.10.10	CLASS1	CO	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
FAILURE	08.11.10	CLASS3	GVHD/AGE	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
INJURY	14.11.10	CLASS2	CO	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	yes	27.11.2010	no	.
RISK	30.12.10	CLASS1	IDIOPATHIC	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
RISK	23.12.10	CLASS1	CO	.	.	.	.	.	persistant rer	no	.	no	.
INJURY	11.01.11	CLASS2	SIRS	.	.	.	.	.	persistant rer	no	.	no	.
INJURY	18.12.10	CLASS2	SIRS	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	yes	24.01.2011
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	normal renal	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	recovered	no	.	no	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
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.	.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	07.03.11	CLASS1	SIRS	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
INJURY	31.01.11	CLASS2	SIRS	.	.	.	.	.	.	.	.	.	.
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RISK	28.05.11	CLASS1	IDIOPATHIC	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
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.	.	.	.	.	.	.	.	.	.	.	.	.
FAILURE	18.04.11	CLASS3	SIRS	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
FAILURE	11.07.11	CLASS3	IDIOPATHIC	.	.	.	.	.	.	.	.	.
INJURY	07.04.11	CLASS2	IDIOPATHIC	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	11.04.11	CLASS1	CO	16.08.11	16.08.11	RISK	CLASS1	CO	.	.	.	.
.	.	.	.	.	.	.	.	.	.	yes	08.04.2011	.
.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	04.05.11	CLASS1	CO	29.05.11	29.05.11	INJURY	CLASS2	SIRS	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
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.	.	.	.	.	.	.	.	.	.	.	.	.
INJURY	09.06.11	CLASS2	IDIOPATHIC	14.08.11	14.08.11	INJURY	CLASS2	IDIOPATHIC	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	yes	06.09.2011	.
RISK	09.02.11	CLASS1	CO	.	.	.	.	.	.	.	.	.
RISK	30.06.11	CLASS1	SIRS	.	.	.	.	.	.	.	.	.
INJURY	18.07.11	CLASS2	NEPHROTOXI	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	yes	24.05.2011
.	.	.	.	.	.	.	.	.	.	.	.	.

.	.	.	.	.	.	.	.	.	.	yes	04.07.2011	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
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RISK	12.09.11	CLASS1	SIRS	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
INJURY	07.09.11	CLASS2	CO	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
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.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	27.02.11	CLASS1	IDIOPATHIC	.	.	.	.	.	.	.	.	.	.
FAILURE	19.08.11	CLASS3	SIRS	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
FAILURE	27.08.11	CLASS3	GVHD/AGE	04.09.11	04.09.11	INJURY	CLASS2	SIRS	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	20.09.11	CLASS1	SIRS	10.10.11	10.10.11	INJURY	CLASS2	.	.	.	.	.	.
INJURY	21.06.11	CLASS2	IDIOPATHIC	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
INJURY	06.11.11	CLASS2	CO	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
INJURY	16.01.11	CLASS2	IDIOPATHIC	.	.	.	.	.	.	yes	25.11.2011	.	.

.	.	.	.	.	.	.	.	.	.	.	.	.	.
INJURY	15.10.11	CLASS2	GVHD/AGE	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
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.	.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	22.12.11	CLASS1	CO	.	.	.	.	.	.	.	.	.	.
INJURY	04.12.11	CLASS2	IDIOPATHIC	28.12.11	28.12.11	INJURY	CLASS2	IDIOPATHIC	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	07.05.11	CLASS1	IDIOPATHIC	27.08.12	27.08.12	INJURY	CLASS2	SIRS	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	18.11.11	CLASS1	NEPHROTOXI	24.05.11	24.05.11	RISK	CLASS1	IDIOPATHIC	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	30.11.11	CLASS1	CO	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
RISK	01.12.11	CLASS1	NEPHROTOXI	18.12.11	18.12.11	INJURY	CLASS2	SIRS	.	.	.	.	.
RISK	31.12.11	CLASS1	SIRS	.	.	.	.	.	.	.	.	.	.
RISK	26.12.11	CLASS1	SIRS	.	.	.	.	.	.	.	.	.	.
INJURY	23.02.11	CLASS2	CO	.	.	.	.	.	.	.	.	.	.
INJURY	15.01.12	CLASS2	CO	.	.	.	.	.	.	.	.	.	.
RISK	18.02.11	CLASS1	CO	23.07.11	23.07.11	INJURY	CLASS2	SIRS	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
INJURY	22.12.11	CLASS2	SIRS	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	.	.	.	.	.
INJURY	10.02.12	CLASS2	NEPHROTOXI	15.03.12	15.03.12	INJURY	CLASS2	NEPHROTOXI	.	.	.	.	.
INJURY	23.01.11	CLASS2	CO	.	.	.	.	.	.	.	.	.	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	no	.
.	.	.	.	.	.	.	.	.	persistant rer	no	.	yes	16.04.2010

CKDyesno	CKD1	CKD2	CKD3	Cr3month	Cr6month	Cr12month
1	.	.	.	0.9	0.7	0.9
1	.	.	.	0.5	0.7	0.5
1	.	.	.	0.6	0.7	0.8
.	.	.	1	.	0.9	0.8	0.9
1	.	.	.	0.4	0.4	0.5
1	.	.	.	.	.	.
1	.	.	.	0.8	.	.
1	.	.	.	0.5	.	.
.	.	.	1	.	1.6	0.8	.
.	.	.	.	1	1.8	1.9	1.9
.	.	.	.	1	1.7	1.5	1.4
.	.	1	.	.	1.2	0.9	0.8
1	.	.	.	0.8	0.7	0.6
.	.	1	.	.	0.8	0.8	.
.	.	.	.	1	1.4	1	1.4
1	.	.	.	0.9	1.1	0.7
.	.	.	1	.	1	1.3	1.1
.	.	.	.	1	1.2	1.3	1.5
1	.	.	.	0.4	0.5	0.5
1	.	.	.	1	0.9	0.9
1	.	.	.	2.4	.	.
.	.	.	1	.	0.8	1	1
.	.	.	.	1	1.5	1.1	1
1	.	.	.	0.5	0.6	0.78
1	.	.	.	0.7	0.6	0.6
.	.	.	.	1	0.9	1.1	1.3
1	.	.	.	0.6	0.5	0.5
.	.	.	1	.	0.7	0.9	1.1
.	.	.	.	1	1	0.9	1.2
1	.	.	.	.	.	.
1	.	.	.	0.5	0.5	0.6
1	.	.	.	0.6	1	0.7
1	.	.	.	0.9	0.9	0.7
.	.	.	1	.	1.2	0.9	0.9

1	.	.	.	1.3	.	.
.	.	.	1	1.1	1.3	1.4
1	.	.	.	0.5	0.4	0.6
.	.	.	1	1.2	1.4	1.5
1	.	.	.	0.7	0.7	0.7
1	.	.	.	1.1	0.7	0.6
1	.	.	.	.	.	.
1	.	.	.	0.9	0.8	.
.	.	.	1	0.9	1.3	1.2
1	.	.	.	2.1	.	.
1	.	.	.	0.7	0.8	0.8
1	.	.	.	3.5	.	.
1	.	.	.	0.9	0.7	0.8
1	.	.	.	0.7	.	.
.	1	.	.	1.2	1.1	0.7
1	.	.	.	0.7	0.6	0.6
1	.	.	.	.	.	.
1	.	.	.	0.5	0.6	0.7
1	.	.	.	.	.	.
1	.	.	.	0.6	0.6	0.6
1	.	.	.	.	.	.
.	.	.	1	1.7	1.3	1.2
1	.	.	.	0.4	0.4	.
1	.	.	.	.	.	.
.	.	.	1	1	1.4	1.7
1	.	.	.	.	.	.
1	.	.	.	.	.	.
1	.	.	.	0.6	0.6	0.5
1	.	.	.	0.8	1	0.7
1	.	.	.	0.6	0.6	0.6
.	.	.	1	1.4	1.3	0.9
1	.	.	.	0.4	0.4	0.5
1	.	.	.	.	.	.
.	.	.	1	1.2	1.2	1.3
1	.	.	.	0.7	0.7	0.8

	1	.	.	.	.	.	.	
.	.		1	.		1.1	1.1	1.1
.	.	.			1	1.2	1.1	1.1
	1	.	.	.		0.6	0.7	0.7
	1	.	.	.		0.7	0.7	0.7
	1	.	.	.	.	.	.	
	1	.	.	.		1.1	1	0.7
	1	.	.	.		0.9	1.1	0.8
.			1	.	.	0.7	0.9	0.9
	1	.	.	.	.	.	.	
	1	.	.	.	.	.	.	
.			1	.	.	0.9	0.8	0.7
.	.			1	.	1.1	1	0.9
.			1	.	.	0.8	0.9	0.9
	1	.	.	.		0.5	0.4	0.7
	1	.	.	.	.	.	.	
	1	.	.	.		0.6	0.5	0.6
.	.	.			1	1	1	1.2
.	.			1	.	0.7	0.9	1
.	.	.			1	1.1	1.3	1.4
	1	.	.	.	.	.	.	
.	.			1	.	0.8	1.2	0.9
	1	.	.	.	.	.	.	
	1	.	.	.	.	.	.	
	1	.	.	.		0.5	0.4	0.5
.	.			1	.	1	1.1	1.1
	1	.	.	.	.	.	.	
	1	.	.	.		0.6	0.6	0.7
	1	.	.	.		0.6	0.6	0.5
	1	.	.	.	.	.	.	
.	.	.			1	1.3	1.2	1.4
	1	.	.	.	.	.	.	
.	.	.			1	1.1	1.3	1.2
	1	.	.	.		0.7	0.6	0.6
	1	.	.	.	.	.	.	

1	.	.	.	0.7	1.2	0.6
.	.	.	1	1	1.1	1.4
1	.	.	.	0.9	0.9	0.8
1	.	.	.	0.6	0.7	.
.	.	1	.	0.6	.	.
.	.	1	.	1.2	1.1	0.9
1	.	.	.	.	.	.
1	.	.	.	.	.	.
1	.	.	.	0.7	.	0.6
1	.	.	.	.	.	.
1	.	.	.	0.5	0.4	0.5
1	.	.	.	0.7	0.8	0.6
.	.	1	.	1.2	1.2	.
1	.	.	.	0.9	1.2	0.9
1	.	.	.	.	.	.
.	.	1	.	1.4	1.2	1.5
.	.	.	1	1.5	1.9	.
1	.	.	.	.	.	.
1	.	.	.	0.6	0.5	0.6
1	.	.	.	.	.	.
1	.	.	.	0.7	0.7	0.6
1	.	.	.	.	.	.
.	.	.	1	1.5	1.4	1.1
.	1	.	.	0.8	0.7	0.7
1	.	.	.	0.6	0.5	0.6
1	.	.	.	.	.	.
1	.	.	.	0.9	0.8	0.9
1	.	.	.	.	.	.
.	.	.	1	0.9	1	1.1
1	.	.	.	0.7	0.8	.
.	.	1	.	1.3	1	1
1	.	.	.	.	.	.
1	.	.	.	0.7	0.6	0.7
1	.	.	.	.	.	.
.	.	1	.	0.9	0.9	0.9

	1	.	.	.		0.8	0.7	0.7
	1	.	.	.		0.8	0.9	0.7
	1	.	.	.		0.5	0.5	.
	1	.	.	.		0.6	.	.
	1	.	.	.		0.4	0.4	0.5
	1	.	.	.	.	.	.	
	1	.	.	.	.	.	.	
.		1	.	.		0.8	0.8	0.7
	1	.	.	.	.	.	.	
	1	.	.	.		0.7	0.8	0.7
	1	.	.	.		0.7	0.5	0.5
	1	.	.	.		0.6	0.4	1
	1	.	.	.		0.5	0.6	0.6
	1	.	.	.		0.4	0.6	0.5
	1	.	.	.		0.6	.	.
.	.		1	.		1.2	0.9	1
	1	.	.	.		0.7	0.5	0.6
	1	.	.	.		0.5	0.4	0.7
.	.		1	.		0.7	0.9	0.9
.	.		1	.		0.9	0.8	1.1
	1	.	.	.		0.4	.	.
	1	.	.	.		0.3	0.3	.
.	.	.			1	1	1.3	1.2
.	.		1	.		1	1	1
	1	.	.	.	.	.	.	
	1	.	.	.	.	.	.	
	1	.	.	.		0.5	0.6	0.7
	1	.	.	.		0.9	0.9	0.8
	1	.	.	.	.	.	.	
	1	.	.	.	.	.	.	
.	.		1	.		1.3	1.5	1
	1	.	.	.		0.8	0.7	0.6
	1	.	.	.	.	.	.	
	1	.	.	.		0.6	0.6	0.5
.	.	.			1	1.1	1	1.1

1	.	.	.	0.6	.	.
.	.	.	1	1.1	1.1	1.2
.	.	1	.	0.9	.	.
.	.	1	.	0.9	0.8	0.9
1	.	.	.	.	.	.
1	.	.	.	0.5	0.5	0.5
1	.	.	.	0.6	0.5	0.6
1	.	.	.	0.7	0.8	0.7
1	.	.	.	.	.	.
1	.	.	.	.	.	.
.	.	.	1	0.7	1	1.5
1	.	.	.	0.6	0.6	0.6
1	.	.	.	.	.	.
1	.	.	.	0.5	0.6	0.6
1	.	.	.	.	.	.
.	.	.	1	1.3	1.2	1.3
.	.	.	1	1.8	1	0.9
1	.	.	.	0.4	.	.
1	.	.	.	0.6	.	.
.	1	.	.	1.2	0.9	0.9
.	1	.	.	0.8	.	.
.	.	.	1	1	0.8	1.4
1	.	.	.	.	.	.
1	.	.	.	.	.	.
1	.	.	.	0.7	0.7	.
.	1	.	.	0.8	0.7	0.7
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a ACUTE KIDNEY INJURY IN ALLOGENIC HAEMATOPOETIC STEM CELL TRANSPLANTATION A dissertation submitted to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfillment of the University regulations for the award of D . M . (B r a n c h – I I I) (N e p h r o l o g y) DEPARTMENT OF NEPHROLOGY CHRISTIAN MEDICAL COLLEGE, VELLORE b BONAFIDE CERTIFICATE This is to certify that the work presented in this dissertation titled “Acute Kidney Injury in Haematopoetic Stem Cell Transplantation” done towards fulfillment of the requirements of the Tamil Nadu Dr. M.G.R. Medical University, Chennai for the D.M. (Branch–III) (Nephrology) exams to be conducted in August 2013, is a bonafide work of Dr...